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EDEMA AND LYMPHEDEMA OF THE LOWER EXTREMITIES*

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Edematous swelling of the lower extremities has attracted a minimum of attention in recent years. The literature on the subject is sparse and can be attributed to a few men, E. V. Allen, R. K. Ghormley, H. C. Hoffman, John Homans, Pratt, Geza de Takats, Servelle and Matthew H. Envoy. A discussion on the treatment of chronic edema of the leg appeared in the Proceedings of the Royal Society of Medicine of London, 1950, Volume 43. Sir Archibald McIndoe, A. M. Boyd, Ian Aird, Sir Harold Gillies and others took part in this symposium. An article by the present author, entitled "Lymphedema of the Extremities", was published in the *Virginia Medical Monthly* of June, 1939. It must be confessed that, of the three vascular systems of the lower extremities, arterial, venous and lymphatic, progress in our knowledge has been least in the lymphatic system.

FORMATION OF TISSUE FLUID

Edema refers to increased fluid within the tissue spaces. Lymphedema refers to increased fluid within the tissue spaces and the lymphatic vessels as a result of inadequate function of the lymphatic system. The source of tissue fluid is the blood vascular system. The osmotic pressure of the blood, maintained by the blood crystalloid and colloid, tends to keep the blood volume intact. It is necessary, however, for elements of the blood to reach the interstitial spaces to carry nutrition to the extra-vascular tissues. Therefore, as the result of arterial pressure, filtration occurs from the blood capillaries into the tissue spaces. This tissue fluid is reabsorbed by two mechanisms, one venous and the other lymphatic. At the venous end of the capillary loop, there is a marked reduction in pressure so that tissue fluid re-enters the blood capillary. Fluid not re-absorbed by the blood capillary enters the lymphatic circulation to be transported back into the blood system through the thoracic duct. The continuous local circulation of the tissue fluid, out of the arterial end of the capillary and into the venous end, is the most important mechanism. Nevertheless, any disturbance of the delicately balanced lymphatic circulation will cause leg swelling. Normally a fairly constant amount of tissue fluid is maintained by the following factors:

1. Blood capillary pressure
2. Colloid (osmotic) pressure of the blood
3. The tissue tension
4. The interstitial fluid pressure
5. The lymph capillaries

FACTORS FAVORING EDEMA FORMATION

Factors favoring edema formation are listed in Table 1.

TABLE I

1. Increased capillary blood pressure (venous stasis).
2. Low osmotic colloid pressure of the blood.
3. Increased blood capillary permeability.
4. Failure of the kidneys to excrete normal amounts of water and salt.
5. Failure of the lymphatic circulation.
6. Low tissue pressure, the fatty pads about the ankle, and fat in general—lipedema and lipodystrophy.
7. Warm environment—heat edema. (Due to increased capillary permeability.)
8. Dependency—postural edema.
9. Immobility—hypostatic edema. (The motor of the leg circulation in motion.)
10. High fluid intake (increases edema if salt is available).
11. High salt intake (increases edema if water is available).
12. Disturbed innervation—the unilateral edema of hemiplegia, tropho-edema, angioneurotic-edema.
13. Constricting bands, wrongly applied elastic bandages, tight garters, supportless shoes, ankle-straps.
14. Anemia.
15. Endocrine disturbances — myxedema, pre-menstrual

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edema, hyperfunctioning tumors of the adrenal gland.
16. Drug administration—ACTH, cortisone.
17. Vitamin insufficiency—Beri-Beri.

Increased capillary blood pressure occurs principally from increased venous pressure as in cardiac failure, venous obstruction, venous incompetency as in varicose veins or in the post-thrombophlebitic state. An increased venous pressure results in decreased absorption of fluid from the tissue spaces; one would expect all venous disturbances to cause marked edema. Clinically, however, disturbances of the veins alone rarely contribute significantly to leg swelling. One frequently sees enormous varicosities and no leg edema, and even if edema is present the removal of the varicosities rarely lessens the edema. On the other hand, the valvular incompetency of the deep venous system that follows ilio-femoral thrombophlebitis is frequently accompanied by leg swelling. In my opinion, this is due more to lymphatic obstruction than to the venous factor.

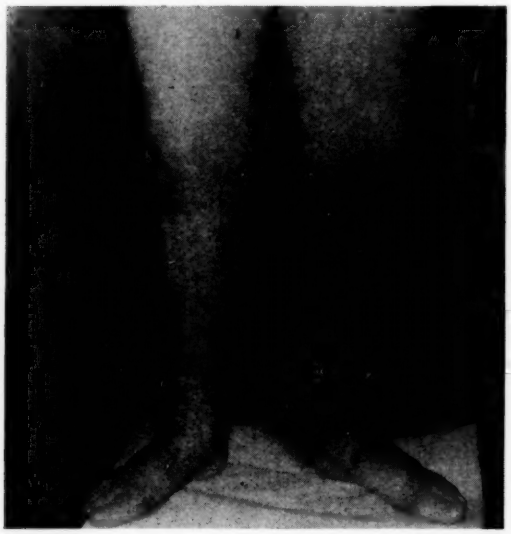

Fig. 1.—Bilateral varicose veins. Moderate edema.

Low colloid osmotic pressure is seen in malnutrition, cachexia of malignancy, nephritis, nephrosis, cirrhosis, large leg ulcers and extensive burns.

Increased capillary permeability is a frequent cause of leg swelling. A local inflammation or contusion or even slight bruise may cause considerable fluid to leave the blood capillaries and enter the tissue spaces. Heat increases blood capillary permeability greatly and explains the frequent occurrence of leg edema during the summer months. Anoxia is a serious cause of increased capillary permeability and explains the irreversible leg edema of advanced peripheral arteriosclerosis.

The lymphatic system is an accessory drainage mechanism which returns colloid and excess tissue fluid to the venous system in the neck when not reabsorbed by the blood capillaries. It is a most delicate system and becomes decompensated extremely easily. Lymphatic circulation failure may be due to congenital inadequacy of the lymphatic system, congenital or acquired lymphangiectasis, valvular incompetency, tubular thrombosis, local, regional or central obstruction.

Many mild cases of leg edema are explained by low tissue pressure. The skin acts as a limiting membrane to inhibit the accumulation of tissue fluid and thin or flabby skin permits leg swelling particularly in hot weather. Fat is a veritable sponge for water. The fatty pads about the ankles are particularly prone to accumulate fluid in the dependent position and obesity accounts for many of the clinical cases of leg edema.

Fig. 2.—Lipedema. Note swelling in fatty pads about ankles.

Even in the horizontal position, the flow of lymph in a motionless extremity is very slight. In the dependent position, there is no lymph flow at all in the immobile extremity. The motor of both the

venous and lymphatic circulation of the lower extremities is motion and immobility and dependency readily cause leg swelling. Add to the picture open, frail shoes or a tight ankle strap and you have the explanation of many swollen feet.

CLINICAL TYPES OF LEG EDEMA

Swelling of the lower extremities may be the initial finding in many and varied conditions. Table 2.

TABLE II

CLINICAL TYPES OF LEG EDEMA

Medical	Vascular	Miscellaneous
Cardiac	Venous Obstruction	Postural
Nephritic	Varicose Vein	Trophic
Nephrotic	Thrombophlebitic	Lipedema
Cirrhotic	Ischemic	Heat Edema
Nutritional	Lymphatic	Traumatic
Endocrine		Inflammatory
Angioneurotic		Mechanical

To classify a given case requires not merely a local examination but a careful appraisal of the patient's history, physical examination and laboratory tests. Special tests are necessary to the understanding of the vascular group-venograms, walking venous pressure tests, twenty-five yard tape test, oscillometric readings.

Not all cases of leg swelling are due to edema. One must exclude lipodystrophy, hemihypertrophy, congenital arterio-venous fistula, acquired arterio-venous fistula, lymphangioma, cavernous hemangioma, arterial aneurysm, sarcoma, Koposi's tumor and bone tumors.

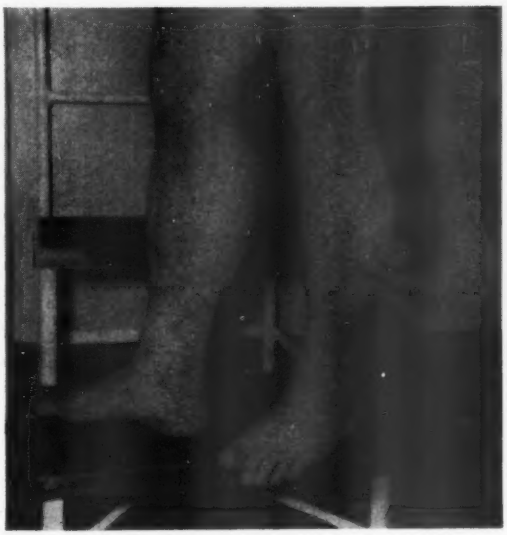

Fig. 3.—Congenital arterio-venous fistula. Erroneously diagnosed as thrombophlebitis.

LYMPHEDEMA

The lymphatic system accounts for many cases of leg swelling. Our knowledge of exactly what pathology exists is practically nil. Clinically, methods of studying lymphatics such as estimation of lymph pressure, x-ray visualization, chemical analysis, and dye visualization have been entirely fruitless. There is some evidence that the radio-active isotopes, particularly yttrium or I-131 tagged serum albumin, may furnish a clinical means of demonstrating lymph flow. The lymphatics are so thread-like in diameter that it requires approximately a magnification of x-8 to visualize them. Moreover, the vessels are entirely transparent because of the colorless lymph and are so thin walled that they have a tendency to collapse during dissection to expose them. A practical approach to the problem of lymphedema awaits further research. It is well to remember that the lymphatic vessels may undergo pathological processes similar to those of veins—dilate, develop valvular incompetency and retrograde flow, develop paradoxical flow from obstruction, thrombose, obliterate from tubular lymphangitis or extrinsic pressure. The lymphatic obstruction may be either in the local lymphatic capillaries, the tubular lymphatics of the thigh, the regional lymph nodes of the groin, or the central lymph nodes of the pelvis or higher.

Fig. 4.—Lymphedema from obstruction of the regional lymph nodes. The patient had granuloma inguinale.

Lymphadenopathy usually means lymphatic ob-

struction and may be due to: 1. non-specific lymphadenitis, 2. lymphoma or metastatic invasion, 3. Hodgkin's disease, and 4. tuberculous lymphadenitis.

DIAGNOSIS OF LYMPHEDEMA

As we do not have definite methods of investigating the lymphatic system, the diagnosis of a given case of leg swelling as lymphedematous must be conjectural. The case is apt to be classified as lymphedema when there is unexplained non-inflammatory simple leg swelling, when the swelling has been present since birth, when the swelling has its onset with puberty, or when there is evidence of malignant disease, surgical removal of the lymph nodes or local x-ray therapy. Inflammation in tubular lymphatics accounts for many cases of clinical lymphedema. Ilio-femoral thrombophlebitis is a common cause of inflammatory lymphedema. The diagnosis of the acute episode is well known. Chronic post-thrombophlebitic lymphedema is diagnosed by the history, the characteristic full feeling in the thigh, the brawny induration and pig skin in the lower leg, the venograms, the walking venous pressure, Scott's twenty-five yard tape test and femoral vein exploration. A classification of lymphedema is presented in Table 3.

TABLE III

CLASSIFICATION OF LYMPHEDEMA

I. Idiopathic or Primary Lymphedema.
II. Non-Inflammatory Lymphedema
  1. Congenital Lymphedema
    a. Simple
    b. Familial (Milroy's disease)
  2. Lymphedema Praecox
  3. Lymphedema secondary to:
    a. Malignant disease
    b. Surgical removal of lymph nodes or interruption of lymph vessels.
    c. Pressure
    d. Roentgen and radium therapy
III. Inflammatory Lymphedema
  1. Traumatic Lymphedema (local tissue injury).
  2. Causalgic Lymphedema (from immobility).
  3. Local inflammation (strep. and staph. infections).
  4. Recurrent Lymphangitis streptogenes.
  5. Post-thrombophlebitic Lymphedema:
    a. From ilio-femoral thrombophlebitis
    b. From repeated episodes of thrombophlebitis
  6. Allergic Lymphedema (Trichophytosis).
  7. Filariasis.

DISCUSSION

Non-inflammatory lymphedema that has no apparent cause is referred to as idiopathic or primary. Unnoticed trauma may be a factor but I am inclined to feel that these are instances of decompensation of the delicately balanced lymphatic circulation. Usually the only history obtainable is that of conversion of a normal limb to a swollen one. The swelling is firm but pits on pressure and it is reduced remarkedly in size when the extremity is elevated. Only in neglected cases do episodes of lymphangitis streptogenes occur.

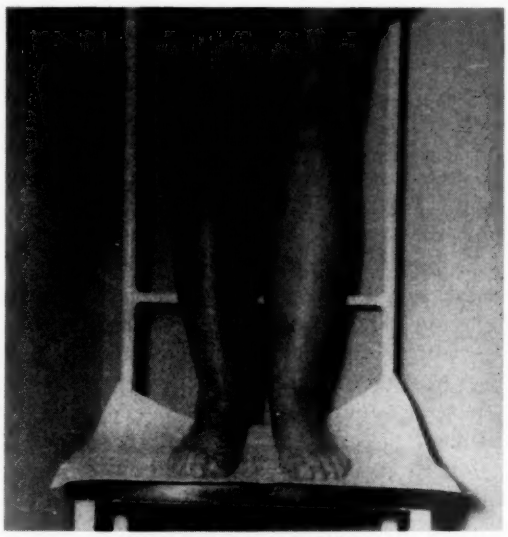

Fig. 5.—Idiopathic lymphedema.

Congenital edema is one of the few types of which we have information concerning the pathology. Tissue biopsy usually reveals greatly dilated thin walled lymph sinuses. A diffuse swelling involving part or all of one extremity has been present since birth. Pain, ulceration or infection rarely complicate the picture. The patients are otherwise in good health.

Lymphedema praecox refers to a non-inflammatory lymphedema that develops at or shortly after puberty. In the majority of cases the onset is between 10 and 25 years of age. One or both extremities may be affected. The edema ordinarily progresses up the leg slowly and the entire limb becomes edematous over a period of months or years. The edema is worse during menstruation and in warm weather, or when the patient is on her feet a long while. The edema is of the pitting variety and disappears on elevation of the extremity. Presumedly there is a traffic jam in the pelvis, the lymph circulation from the pelvic organs blocking drainage from the extremities. However, the actual pathology is unknown.

Lymphedema secondary to malignant disease. Swelling of the leg may be the first indication that the patient has a primary or metastatic malignancy. The swelling may indicate carcinomatous invasion of the lymph vessels, malignant infection of the lymph nodes, or obstruction from peri-lymphatic pressure. In the past several years, we have encountered nineteen instances of leg swelling due to the following causes:

Fig. 6.—Lymphedema praecox. A 12 year old girl whose right leg began to swell at the age of 10.

Cancer of the cervix	10
Cancer of the prostate	2
Sarcoma of the soft tissues of the thigh	2
Cancer of the colon	1
Melanoma of the foot	2
Seminoma of the testis	1
Koposi's tumor	1

One should be especially suspicious of a malignant cause of leg edema when there is persistent pain, progessive edema, only slight improvement with bed rest, no improvement from sympathetic nerve block, and enlarged inguinal nodes. Upper thigh, buttock or scrotal edema, indicating high lymphatic obstruction is to be considered evidence of malignancy until proved otherwise. When a patient has had x-ray therapy for known malignancy and develops leg swelling, it is extremely difficult to arrive at a proper diagnosis. The edema may be due to x-ray fibrosis or recurrence of malignancy.

Traumatic edema is frequently seen clinically. Swelling initiated by a twist of the ankle or a trivial blow on the shin persists indefinitely. Presumedly an inflammatory reaction has occurred resulting in a destruction of the local lymphatics.

Causalgic lymphedema occurs in the painful limb. Because of fear of pain or actual pain the patient fails to move the limb with resultant leg swelling. Inflammatory lymphedema from local infection is familiar to us all. Infected surgical incisions, lacerations, abscesses, furuncles or infected penetrating wounds may all cause inflammatory lymphedema.

Recurrent lymphangitis streptogenes. This is one of the most commonly encountered types of inflammatory lymphedema. The patient is subject to episodes of erysipelas-like attacks which are sudden in onset and striking in severity. Coincident with the development of a small reddened area on the extremity, the patient becomes generally sick with chills and fever. The reddened area spreads until a considerable portion of the extremity is swollen and tender and the skin of the involved part raised and hot. The inguinal lymphadenitis abates and the signs of inflammation disappear. The limb remains swollen, its cutaneous surface discolored and its subcutaneous tissue fibrosed. Unless prophylactic therapy is instituted, attacks follow attacks for years. In addition to the disability caused by the repeated inflammatory episodes, the complications of chronic inflammatory lymphedema, indolent leg ulceration, chronic indurated cellulitis, non-filiarial elephantiasis plague the patient the rest of his life.

Presumably lymphangitis streptogenes is due to an

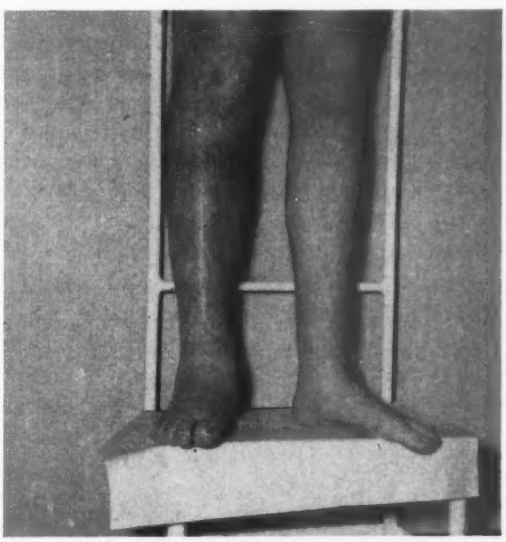

Fig. 7.—Recurrent lymphangitis Streptogenes. The patient has had episodes of chills and fever and leg inflammation for twelve years.

invasion of the subdermal lymphatic by streptococci. The portal of entry for the infection is often a crack in the skin of the foot. Trichophytosis is usually present.

Each attack of lymphangitis streptogenes renders the patient more susceptible to further attacks.

THE TREATMENT OF EDEMA AND LYMPHEDEMA OF THE LOWER EXTREMITIES

The edematous leg should never be passed by lightly. Edema tends to perpetuate and even aggravate edema. A few weeks of uncontrolled edema and a process of fibroblastic proliferation has already begun so that now the leg may well become the chronically indurated irreversibly swollen leg. Furthermore, a persistently edematous leg is extremely susceptible to recurrent infection of the subdermal lymphatics—a condition known as lymphangitis streptogenes. So all acutely edematous legs, whether slightly or greatly swollen, should be treated energetically from the onset while the edema is still reversible. The edema should be controlled for many months until the swelling tendency is overcome. A discussion of the treatment of the more important types of edema and lymphedema follows.

LEG EDEMA (EXCLUSIVE OF LYMPHEDEMA)

Varicose Edema

Peculiarly the eradication of varicosities will relieve only the mildest cases of leg edema. It is important not to promise the patient that fixing his veins will stop his leg swelling. The proper procedure is to perform the usual high saphenous ligation, strip the varicosities to the knee and perform a modified Kondoleon procedure from knee to ankle. The Kondoleon phase of the operation removes the remaining varicosities, interrupts the perforator veins and removes the water-logged subcutaneous tissue.

Ischemic Edema

An extremity that has so little blood supply that it is painful day and night and persistently edematous, will invariably have to come off.

LYMPHEDEMA

Non-Inflammatory Lymphedema:
Congenital Lymphedema

The multiple stage modified Kondoleon procedure, placing normal skin on normal muscle, remains the accepted management.

Adolescent Lymphedema (Lymphedema Praecox)

All we have to offer is an adequate support. The patient must accept the principle of giving her skin a lift with a strong elastic stocking or bandage for life. Otherwise, not only will her condition get worse but she will be subject to recurring episodes of lymphangitis streptogenes. Should the lymphedema pass beyond the pitting stage, a modified Kondoleon procedure may well be performed. Lymphedema secondary to malignant disease rarely responds permanently to treatment. But Roentgen therapy to the invaded lymph nodes may often give temporary relief.

Idiopathic Lymphedema

The condition is no respecter of age but most of our cases have been very young people, often children and usually the female sex. *Case Report:* A 26 year old Navy wife one hot summer day observed that her foot was swollen. Over a period of 15 months, the swelling recurred during the day and receded during the night. The Navy performed a lumbar sympathetic ganglionectomy with no improvement. She has responded well to the constant wearing of a primary bandage over which has been placed elastoplast support. She has consented to an experimental procedure which I hope will be practical for her and other similar cases—that is, the application of an internal skin lift rather than an external support. We will place under the skin,

Fig. 8.—Idiopathic lymphedema of the left foot.

after removing the edematous fatty subcutaneous tissue, a Cutis graft, in the hope that this graft will contract down and stop the edema tendency. Should the swelling involve the leg, we might perform a Kondoleon procedure from knee to ankle and apply a subcutaneous Cutis graft over the foot.

My program for a mild or moderate case of idiopathic lymphedema is as follows: ammonium chloride—grains 15—after meals and at bed time for 5 days a week, followed by mercuhydrin by mouth or by hypodermic for 2 days a week; support of the leg with an Ace bandage or an elastoplast bandage or Unna boot, depending on the difficulty of control of leg swelling, elevation of the foot of the bed 12 inches during the sleeping hours, a rest period during the middle of the day in a similar position, and moderate restriction of fluid intake and salt. As trite as this program seems, it will often restore the lymphatic circulation to compensation so that the patient will be at least temporarily cured. These cases are usually encountered only during the summer months, the increased permeability of the hot days causing decompensation of the lymphatic system. If compensation can be restored by the methods outlined, the cases will remain compensated the rest of the year.

TRICKS OF THE TRADE

Diuresis. Marked diuresis tends to abstract fluid from the tissue space and also increases tremendously lymph flow. This has been proved repeatedly in animals. It is well not to think disparagingly of diuresis in the management of lymphedema. The lymphogogue affect is profound and furnishes us with a sound and practical method of helping the cases under discussion.

Elevation. There are many tricks of the trade in carrying out the measures which improve the lymph flow. Thus, true elevation of lymphedematous leg really means elevating the foot as high as the neck because after all the lymph has to get back into the venous system through the thoracic duct at the neck level. For bed rest, there must be shock blocks under the foot of the bed or a kitchen chair, not simply pillows under the knees. Twelve inches of elevation is average but greater heights may be required. For bedside sitting the leg must be raised above the hip level. A foot stool is inadequate unless especially high. How often does one see a patient rolling around the wards in a wheelchair with markedly edematous feet! If they spill some water on their dinner tray in the same position, one would surely see the water fall to the lower end of the tray. I have restored many legs to lymph compensation by simply raising the legs higher and higher.

Compression. Adequate compression is also often wanting. In the pitting stage, the leg swelling is always reversible. Patients tend to get hold of bandages with no elasticity at all or to continue to use their bandages after the elastic element has gone. When one method of compression fails, another will do the job. If one elastic bandage will not hold down the edema very well, an Unna paste boot from toes to groin will often relieve the edema when bandages have failed. Or the change from an Ace type of bandage to a strong Davol rubber bandage may succeed where other types have failed. One can always squeeze water out of a sponge. When a lymphedematous leg swells in spite of support, it simply means that the support has not been put on early enough in the morning before the tissue spaces became distended. Another explanation is that the support has been too weak.

There are tricks also in obtaining compression where most needed. For a swollen ankle, the insertion of a pad of sponge rubber underneath the elastic support will often rid this area of persistent edema. A simple change from the usual supportless high heeled shoe to an Oxford type may make the difference between failure and success.

Massage. Massage is an excellent method of increasing lymph flow and is entirely safe in the non-inflammatory group under discussion.

Exercise. We often remind the patients that the motor of the lymph circulation is motion and that they must move their feet up and down when they stand still and walk energetically. Another good form of leg exercise consists of bicycling exercises with the legs entirely overhead.

The Cervix. And, lastly, one must not forget that in this group of cases, which are usually girls or young women, there may be a factor of central lymphatic obstruction from infected lymph glands, secondary to cervicitis. Cervicitis should always be corrected in young girls with lymphedema.

ACUTE INFLAMMATORY LYMPHEDEMA

The tubular lymphatics are known to thrombose and obliterate just as do the veins. *Thus the need*

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arises for rapid anti-coagulant therapy in the acute phase of all inflammatory lymphedemas. It is equally important to control leg swelling and to institute promptly measures aimed at restoring the extremity to its normal size. The tissue fluid of acute lymphedema is characterized by its high protein content which acts as a culture medium for fibroblastic proliferation. Each day that the limb is allowed to remain lymphedematous means a greater possibility of permanent lymphedema and eventual fibrosis—the chronically indurated leg.

Traumatic Lymphedema. Bed rest is instituted with the foot of the bed elevated on shock blocks 12 inches high, warm compresses are applied, and rapid anti-coagulant therapy begun. Acute inflammatory lymphedema, as a result of local infection, is handled similarly plus anti-biotic therapy. Occasionally x-ray therapy is used for the slower resolving lesions and the acute regional lymphadenitis.

Acute Lymphangitis Streptogenes. We paint the inflamed skin with 10 per cent silver nitrate, apply voluminous warm compresses of one to ten thousand silver nitrate or boric acid solution, elevate the leg, give anti-biotic therapy in the form of penicillin or Terramycin and as in all inflammatory lymphedemas institute rapid anti-coagulant therapy. *The acute allergic lymphedemas,* such as "Id" reactions from over treatment of a local dermatitis or allergic dermatitis from local fungus infection, respond well to ACTH and Cortisone.

Thrombophlebitic Lymphedema. In the acute stage, the edema is of the lymphedematous type because the proximal tubular lymphatics are obstructed as they course in the distended vascular sheath in the groin. In the chronic stage, the swelling is of the lymphedematous type because the proximal tubular lymphatics are obstructed within the scarred vascular sheath in the groin and the local lymphatics in the lower leg are destroyed by a fibroblastic proliferation. The management of acute iliofemoral thrombophlebitis is fully documented-bed rest with the leg well elevated, voluminous warm compresses from toes to groin, rapid and effective anticoagulant therapy and para-vertebral novocaine blocks of the second, third, and fourth lumbar sympathetic ganglia until the fever subsides and leg swelling disappears. Please note that I have not included anti-biotics, especially penicillin. These cases are rarely of bacterial origin and such therapy is a waste of medicine and money. Prolonged bed rest is contra-indicated. The thrombus heals *in situ* in 48 to 72 hours and, providing anti-coagulant therapy has been energetic, no new thrombi should form. The average case should be ambulatory in 8 to 10 days.

During the convalescent stage, detailed attention is given to avoid the recurrence of leg swelling. The patient continues to sleep with the foot of the bed elevated twelve inches. When sitting, his leg is on a chair plus a pillow so that his foot is almost as high as his clavicle. Nothing is more pernicious than to have the patient riding around in a rolling chair. His leg will invariably be slanting downward and swell again. Adequate compression of the leg is maintained by Ace No. 8 bandages. Lymph flow is stimulated by diuresis. Adequate compression is maintained for 8 months to a year. The patient is given written post-thrombophlebitic directions outlining his new way of life.

TABLE IV

CARE OF THE POST-THROMBOPHLEBITIC LEG*

1. Wear your elastic stocking or elastic bandages from the time you get out of bed until you retire, with the exception of bath time. The stocking should be renewed every three months, and it is best to have two stockings that can be alternated for cleaning purposes. If bandages are used, have several on hand and use a different one each day and be sure that they will have their elasticity.
2. Do not stand for more than thirty minutes without sitting down for fifteen minutes and elevating the leg on another chair. When standing, get into the habit of flexing the toes in your shoes and frequently rising on tip toes.
3. Plan your day so that you can lie down for two to three half hour periods and elevate your leg to a 45 degree angle. The back of a small straight backed chair is useful for this purpose.
4. Whenever you sit down, elevate your leg on a foot stool, chair or sofa. Avoid allowing the leg to dangle motionless.
5. At night raise the foot of the bed on blocks about six inches.
6. Avoid irritation to the involved leg, especially in respect to sunburn.
7. Be extremely careful to prevent bumping, bruising, or scratching the affected leg.
8. Do not drink excess fluids or take excess salt in foods.
9. Remember that the motor of the leg circulation is motion. Raise up and down on the toes 10 times, two or three times a day preferably with shoes off.

EUGENE L. LOWENBERG, M.D.

*Modified instructions of Luke, "The New Way of Life", for the Post-phlebitic Leg, *Canadian Medical Association Journal,* **61**:270-275, 1949.

CHRONIC LYMPHEDEMA AND THE "INDURATED LEG"

The process results from mismanagement of or repeated episodes of traumatic lymphedema, local inflammatory lymphedema, recurrent lymphangitis streptogenes, thrombophlebitis in varicose veins, iliofemoral thrombophlebitis. In fact, any uncontrolled lymphedema—even congenital lymphedema and lymphedema praecox over a period of years—may produce such a leg. Chronic leg ulcer is a frequent concomitant of the legs under discussion.

Fig. 9.—Bilateral post-thrombophlebitic leg ulceration and induration.

Medical Management. There are instances in which the pathology is borderline or the age of the person or other factors contra-indicate surgery. Some evaluation of the opportunities for improvement on medical management can be obtained by noting what per cent of the lymphedema is pitting much and what per cent is hard and irreversible and how such recession of the swelling occurs during bed rest at night. A further important factor is the willingness of the patient and the ability of the patient to lead the post-thrombophlebitic life. Medical management may meet with surprising success in instances of post-thrombophlebitic lymphedema of short duration. Here para-vertebral novocaine blocks of the sympathetic ganglia are remarkedly effective even several months after the acute episode. We usually hospitalize the patient for a few days, elevate the foot of the bed high, apply compression and institute dehydration and diuresis. Usually the leg swelling rapidly subsides. We then perform para-vertebral novocaine blocks once to several times until the residual leg swelling is overcome. The lymphatic circulation has now been restored to compensation and with good care can be so maintained. It must be clear to such a patient that his life is forever one of elastic support for the leg.

Fig. 10.—Six weeks post-operative. The skin, subcutaneous tissue, and fat have been completely removed from each leg and the legs resurfaced with normal skin or normal muscle.

Surgical Management. For a number of years, I have applied a modified Kondoleon procedure for chronic indurated cellulitis. The principles involved are the removal of the lymph soaked subcutaneous tissue and fascia and the resurfacing of the leg with normal skin resting on normal muscle. Any co-existing venous pathology is handled simultaneously by ancillary procedures. It is axiomatic that no matter how horrible a leg appears from venous or lymphatic system pathology, there is a normal leg underneath at the muscle level and with proper surgical management such a leg can be restored to relative normalcy. The surgery to be performed is evaluated by a pre-operative estimation of the limits of the existing damaged skin, the extent of the underlying subcutaneous tissue induration, the presence

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TABLE V

AUTHOR'S SURGICAL PROCEDURES FOR CHRONIC INFLAMMATORY LYMPHEDEMA (THE INDURATED LEG)*

Surgical Procedures	Indications
1. Simple removal of the indurated subcutaneous tissue and fascia by a modified Kondoleon procedure.	1. When the existing skin is normal and no serious venous pathology exists.
2. High saphenous ligation and a stripping procedure to knee with a modified Kondoleon procedure from knee to groin including removing the great saphenous system and perforators from knee to groin.	2. Where varicosities complicate the picture.
3. A saphenous ligation and stripping, a superficial femoral or popliteal ligation, a Kondoleon-like procedure.	3. For the post-thrombophlebitic state.
4. The same procedure plus excision of all damaged skin plus resurfacing the leg with a split thickness skin graft placed on normal muscle.	4. Where skin pathology exists.
5. The same procedure plus excision of the ulcer, its underlying bed and fascia.	5. Where post-thrombophlebitic ulceration exists.
6. The same procedures plus lumbar sympathectomy.	6. For indications previously mentioned, and causalgia, excessive sweating, and ischemia.

*The Surgical Management of Chronic Indurated Cellulitis of the Lower Extremity (the Indurated Leg), *Surgery*, **28**:832-850, 1950.

of varicose veins, the evidence of an incompetent recanalized femoral vein, the presence of an ulcer, and, finally, the evidences of arterial spasm such as coolness and sweating. The surgical procedures employed are outlined in Table V. The residents call procedures 3, 4, and 5 "All American," and I guess rightly. The procedures are radical and there is a lot to be done. They require a good working team but the results are excellent. All existing pathology is effectively corrected and a chronic invalid is restored to relatively normal life.

Superficial Femoral (or Popliteal) Vein Ligation and Lumbar Sympathectomy. Some authors maintain that superficial femoral or popliteal ligation alone will improve the lymphedema of this type of leg. With this I entirely disagree. I do not believe that any type of removal or ligation of veins will effectively help leg swelling. I include the deep vein ligation in my "All American" procedures for two reasons: one, to stop the venostasis in the erect position, and, two, to lessen the chance of post-operative pulmonary embolism.

The place of lumbar sympathectomy has similarly been somewhat controversial. Lumbar sympathectomy alone will not do anything for post-thrombophlebitic leg swelling that one or several paravertebral novocaine blocks will not do. I know of no type of lymphedema in which you can promise the patient that the leg swelling will disappear after a lumbar sympathectomy. It does have its place but only for specific indications such as causalgia, increased moisture, arterial insufficiency.

SUMMARY AND CONCLUSIONS

1. The edematous leg should never be passed by lightly, for edema tends to perpetuate and even aggravate edema. All acutely edematous legs should be treated energetically from the very onset.
2. Eradication of varicosities will only relieve the mildest cases of associated edema. When considerable edema exists, vein ligation and stripping should be accompanied by a Kondoleon procedure on the lower leg.
3. The lymphedema praecox case, treated nonsurgically, must accept the principle of giving her skin a lift for life by wearing a strong elastic support.
4. The lymphatic system of the lower extremities has practically no reserve capacity and readily becomes decompensated. Many cases of idiopathic lymphedema can be so explained. Measures which act as lymphogogue or otherwise tend to restore lymphatic compensation are (1) elevation, (2) compression, (3) exercise, (4) massage, (5) restriction of salt and water, and (6) vigorous diuresis.
5. Diuresis is a true and powerful lymphogogue—not merely a dehydrating process. It is well not to look too disparagingly upon diuresis in the management of mild lymphedema.
6. There are many tricks of the trade in restoring a limb to the non-edematous state: (1)

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higher elevation of the part, (2) stronger compression, (3) sponge pressure, (4) increased active leg exercises, (5) a change to shoes offering greater foot support.

7. Mild unexplained lymphedema in women may be secondary to cervicitis. Ascending infection of the pelvic lymph nodes causes central lymphatic obstruction of the leg lymphatics. In such instances, clearing the cervix may cure the lymphedema.
8. Vigorous anti-coagulant therapy is an essential part of the treatment of all types of inflammatory lymphedema—to limit the destruction

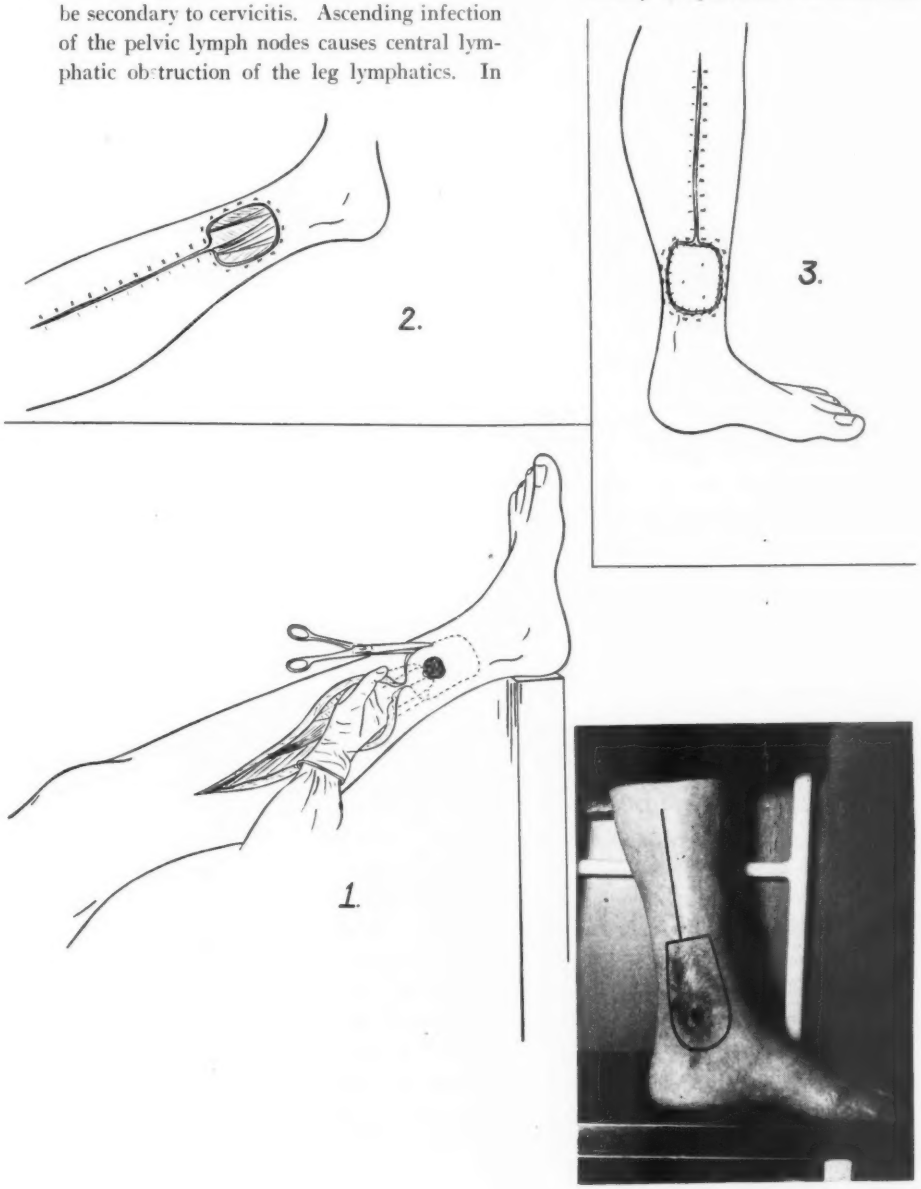

Fig. 11.—Surgical management of chronic lymphedema and leg ulceration. Reprinted from Surgery 28:832-850, 1950. Lowenberg, "The Surgical Management of Chronic Indurated Cellulitis of the Lower Extremity (the Indurated Leg)."

by thrombosis of the lymphatic channels.

9. Acute allergic lymphedema responds well to ACTH and Cortisone.
10. The leg swelling of acute ilio-femoral thrombophlebitis is lymphedematous. The mainstays of treatment are anti-coagulant therapy and paravertebral blocks of the lumbar sympathetic ganglia. Prolonged bed rest is contra-indicated. Penicillin has no value.
11. The patient who has had ilio-femoral thrombophlebitis should lead a "post-thrombophlebitic life" for 8 months to a year. Should lymphatic circulation decompensation occur several weeks or even several months after the acute episode, vigorous medical measures will restore lymph circulation compensation and relieve the lymphedema. Para-vertebral novocaine blocks of the lumbar sympathetic ganglia are effective measures in the medical management even at this late date.
12. Lumbar sympathectomy is of limited value in chronic lymphedema. Simple para-vertebral novocaine blocks will do as much for the leg swelling. But lumbar sympathectomy is indicated for causalgia, excessive perspiration, and ischemia.
13. When chronic inflammatory lymphedema has produced the "indurated leg" only surgical measures will effectively help the patient. We perform several types of composite one stage operations, most of which have as their basis the removal of all pathologic skin, subcutaneous tissue, fascia, the simultaneous correction of existing venous pathology, and the resurfacing of the leg with normal skin on normal muscle.
14. Superficial femoral vein or popliteal vein ligations alone will not cure post-thrombophlebitic lymphedema. We include these measures in our composite operations only to relieve venous stasis and to prevent pulmonary embolism.
15. No matter how horrible a leg appears from venous or lymphatic pathology, there is a normal leg underneath—at the muscle level. With proper surgical management such a leg can be restored to relative normalcy.

100 Medical Arts Building.

New Interns Appointed.

In the past the short supply of new interns caused much confusion in attempts of students and hospitals to get together. Some hospitals exerted a variety of pressures to obtain the interns they needed. Students, anxious not to be left out of choice positions, frequently made unwise or hasty decisions.

In an article in the May 1952 issue of *The Journal of Medical Education,* Dr. F. H. Mullin, Dean of Chicago Medical School and chairman of the internship committee and John M. Stalnaker, director of studies for the Association of American Medical Colleges, report on the results of the first year's matching. They express the opinion that the plan may be even more fully utilized when its success is generally known.

Mr. Stalnaker quotes the dean of one medical school, who said, "Never before in my 17 years of experience have internship appointments been handled so smoothly."

In March of this year punched rating cards were run through a machine which automatically matched hospitals and interns, in most cases with their first choices.

Approximately 95 per cent of this year's interns participated in the plan. Some 98 per cent of the hospitals offering internships were participants.

The National Interassociation Committee on Internships is composed of representatives from the Association of American Medical Colleges, the Council on Medical Education and Hospitals of the American Medical Association, the American Hospital Association, the American Protestant Hospital Association, the Catholic Hospital Association and the medical services of the Federal agencies offering internships.

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SUBTYPES OF MIGRAINE*

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Migraine is a term which applies to a type of recurrent headache. It appears at variable intervals, is apt to appear in several members of a family, is frequently preceded by visual disturbances and accompanied by nausea and vomiting. It is almost impossible to offer an entirely satisfactory definition of migraine as there is no general agreement about the scope of the term. Some include in migraine all cases of chronic recurrent headache, whereas others confine the term to the pattern of headache just described. The latter group is a relatively smaller group in which there is tendency to hemicrania preceded in most instances by a prodrome or an aura and being accompanied at the conclusion of the headache by nausea and vomiting. Insufficient attention, however, has been drawn to the many cases of migraine which depart from the conventional or classical pattern described in most textbooks. They are frequently mentioned as variants or equivalents or unusual types. Yet in our experience they are almost as common as the conventional type which is preceded by visual disturbances.

These symptomatic departures from classical migraine are frequently not recognized as part of the migraine symptom complex because other phenomena intervene and make for confusion and faulty interpretation of the disorder. The relative frequency of these variants and subtypes is almost as common as classical migraine and warrants more complete description. The material which formed the basis of this paper was collected in less than a year from a series of cases who were seen because of recurrent headache.

By way of short review, it is well to remember that migraine is a pattern of dysfunction within the nervous system manifested as mood and body disturbances both painful and non-painful. The typical migraine attack is only one aspect of a diffuse disturbance of function occurring periodically. Other bodily disturbances may be accentuated and become the basis of the major complaint, with reduction or accentuation of preheadache and headache phenomena.

The pathological physiology concerned in the migraine syndrome is practically universally agreed upon as being a wave of initial local vasospasm of cerebral arteries or of retinal vessels followed by vasodilator manifestations. Electroencephalogram recordings from the visual cortex, in two instances, were reported by Engel and his associates as showing transient focal abnormalities in the e.e.g. tracings corresponding to the location and duration of the focal neurological signs. These cases were individuals who had typical scintillating scotoma, hemianopsia and hemicrania. These findings are strong in support of evidence that migraine reactions are mediated through vascular mechanisms. The circulatory phenomena act as temporary irritant or paralyzing agents on the functions of cerebral areas. The two mechanisms may overlap but usually there is a free interval before headache appears. The possible clinical pictures and entire clinical picture is more readily understood if one keeps in mind the fact that the disturbance of varying cortical and subcortical areas for specialized function are responsible for the symptoms noted.

As already mentioned, a very common type of migraine is that in which there is vasospasm or vasodilating phenomena progressing from the occipital lobe with scotoma, flickering or flashings of light, revolving circles, display of colored lights or fortification spectra. These are referable to one or the other occipital lobe and hemianopia in the corresponding visual field develops and precedes the typical hemicrania type of headache. If one considers this same type of disturbance of function arising in other areas of the brain the clinical expression has a wide scope of symptom possibilities that depart from this pattern of headache and it is with these types that we wish to concern ourselves in this discussion.

If vasospasm or dilation involves the parietal lobe there will be resulting paresthesia in the nature of numbness, pins and needles sensation, or reduction of sensation of parts of the opposite side of the

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body. These individuals then have a prodrome or an aura in which the first manifestation is not visual but is one of subjective sensory disturbances, frequently about the face, cheek or in the fingers. The conventional pattern then follows in which the sensory disturbance may extend to one entire side or it may remain localized, then subsides and after a short free interval of 20 to 45 minutes is followed by typical hemicrania, nausea and vomiting.

In other instances the motor area is the first to become affected with a resulting transient paralysis, usually of an arm, lower face, and in one of our patients, a complete hemiplegia.

When the spread of the mechanism becomes generalized or involves areas for specialized functions, such as consciousness, speech, motor skill, taste, hearing, coordination, and of individual cranial nerve mechanisms, a wide variety of confusing symptoms and signs can appear. Loss of consciousness, clouding of consciousness, fugue like states may appear if thalamic and hypothalamic mechanisms are involved. One of our cases, seen recently, gave a history of migraine headaches ever since the age of onset of menstruation. With the appearance of her menopause ten years ago, her headaches changed to periodic seizures of another character. She began to have episodes of dizziness, unsteadiness in walking, dysarthria and aphasia that were very short in duration. In some of these there would be sudden loss of consciousness—up to 20 or 30 minutes—without convulsion, then unsteadiness in walking and some speech disturbance. These attacks have been recurring for ten years and the migraine element of headache has become less intense and less frequent. Hypothalamus participation in the migraine attack is also demonstrated by the hyperpyrexia and unexplained fever that frequently is part of the migraine seizure. Localization of the point of origin of almost all of the signs and symptoms can be made. When opthalmoplegia occurs as the preliminary or preheadache phenomenon the disturbances implicate the midbrain or mesencephalon. Recurrent cranial nerve paralysis, particularly those supplying ocular muscles, are seen. We have observed the recurrent appearance of Bell's palsy in two sisters, both of them afflicted with migraine. One other case has had 7 attacks of Bell's palsy of transient character in association with a migraine attack. Many of these cranial nerve palsies remain for several days after the headache subsides. One of the sisters mentioned above now has a residual Bell's palsy. Facial neuralgias in the trigeminal area frequently accompany the headache and we have seen one patient whose preheadache symptoms included severe pain in the roof of the mouth, palate and pharynx on one side. She suffered typical migraine attacks from the age of 15 to 25, then had a quiescent period and then her headaches recurred 3 years ago, with a prodrome and aura consisting of throbbing in her head, buzzing ear sounds, pain in roof of mouth, palate, left side of nose and forehead, the pain coming on in severe waves every few hours, headache being constant. After bouts of headache she had a peculiar sensation of euphoria and excessive appetite. Tinnitus, roaring head noises, hissing sounds and gustatory phenomena ushered in the headache in other cases. Extreme vertigo with Meniere's like symptoms has been seen in a number of cases with typical migraine headache following. Vomiting in several patients initiated the attack but in most instances seemed to terminate the attack and implies that the medulla takes part in the vasomotor disturbance.

Temporal lobe localization is suggested by those cases having auditory phenomena and gustatory phenomena as aura and is also suggested by those cases in which there is receptive type of aphasia, the patient in the pre-headache phase being unable to either grasp what he hears or is unable to express himself adequately. Aphasia, alexia, agraphia of transient character have also been observed. Memory and recall are affected and are sometimes referred to as holes in the memory.

When the vasomotor disturbance arises in the region of the angular gyrus, a region between the parietal and occipital cortex, patients may have a very disturbing type of visual manifestation. In this instance the individual has as preheadache aura or phenomena a type of spatial disorientation, inability to properly orient himself in his immediate surroundings or to the primary directions, with confusion of right and left and defective appreciation of depth perception.

Cerebellar symptoms, such as ataxia, nystagmus, disturbance of equilibrium, have been very disturbing in two of our patients and in both instances appeared after the headache had developed. In another case the marked light-headedness, cerebellar ataxia in walking and blurring of vision occurred for

two days preceding each typical attack of migraine. More recently this lady developed loss of consciousness for ten-fifteen minutes in her attacks, this being followed by diplopia, slurred speech and marked loss of coordination and severe headache requiring opiates for relief. The entire attack often lasted for two or three days. Migraine as a child was typical—hemicrania, occipital to start with, and then involved one-half of head.

Fugues and psychomotor phenomena are very disturbing in some individuals, particularly if they occur in the preheadache period. We have observed several instances of this character. They are important for the psychiatrist to recognize and difficult for the neurologist to differentiate from psychomotor epilepsy. The fugues appear suddenly, precede or accompany the headache. They resemble the psychomotor attack of the epileptic but are never characterized by convulsion or by acts of violence or sadistic behavior. Usually the individual acts dull and stupid and rambles in speech in an inarticulate or irrelevant manner.

In some individuals there seems to be a parallelism or correlation with convulsions. The migraine-epilepsy relationship has frequently been referred to in literature and sometimes is called migraine epilepsy. Actually the number of such cases reported is relatively small. We have had a number of experiences where epileptics have gained control of their seizures, but where the seizures are still represented by recurrent, periodic migraine attacks which were then controlled by adding to the dosage of the anticonvulsant drug.

Recently attention has been called to abdominal migraine and abdominal epilepsy. The dominant preheadache aura and symptoms in these individuals are discomforts and pains associated with thoracic, abdominal or pelvic organs and cavities. Fitz Hugh noted precordial migraine or pain in 27 cases of a series of 880 patients with migraine. Attacks of tachycardia, dyspnea, precordial pain, epigastric distress or abdominal pain frequently associated with nausea and vomiting may be predecessors or accompaniments of the migraine attack.

In children these bouts of abdominal pain, nausea, vomiting and headache commonly terminate in sleep, following which the patient awakens feeling quite well. We and others reported a series of such cases admitted to a pediatric service for observation as surgical abdominal conditions. One series of about 9 cases all had this pattern, had suspicious e.e.g. changes of epilepsy and 7 cases, within the next ten years, did develop epilepsy.

Urticaria, erythema, pallor, angioneurotic edema, watery nasal discharge, salivation, unilateral hyperhidrosis, and scleral congestion may accompany the migraine attack and imply involvement of the autonomic system just as the visceral neuralgias do or that the cortical representation for these functions has been irritated. Oppenheim described finding conjunctival and retinal hemorrhages and also reported the periodic occurrence of nosebleed with the headaches.

A close relationship has been observed in children between cyclic vomiting in infancy and periodic headache appearing later in childhood. In several of our cases the one condition seemed to have gradually replaced the other, the cyclic vomiter of childhood becoming the sufferer from classical migraine in childhood or adolescence.

Time does not permit us to recite other specific instances of the various symptom complexes encountered in the migraine picture which result in symptomatic departures. We do wish to call attention, however, to interesting interparoxysmal symptoms which many of the migraine sufferers may have between their attacks. There are tendencies to neuralgias, vertigo, tinnitus, psychic depression, gastralgia and also attacks of extremely sudden increased intestinal peristalsis with explosive bowel movements, spasms of yawning and sometimes sneezing. These are noted particularly in the older, longstanding cases.

The symptomatic range of migraine is therefore very wide and includes the interparoxysmal period. Rudimentary forms are at times hard to recognize as one or more than one of the characteristic classical migraine syndrome symptoms may be lacking. Severe cases can simulate cerebral disorder of a more serious kind, particularly cerebral thrombosis and multiple sclerosis. Others are frequently considered to be hysterical. It is worthy of note that many cases of migraine occur without the conventional onset with visual aura. Migraine is a disturbance of diffuse and local cerebral function, the areas involved being responsible for the particular presenting symptom. In our opinion any portion of the cerebral structures and integrated functions can take part and

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it is our belief that this has not been adequately and fully emphasized.

Caution must be exercised in these subtypes or variants that one is not dealing with a symptomatic form of migraine in which typical migrainous seizures may appear, for a time, as the presenting and only manifestation of some definite disease of the brain or its coverings, notably intracranial neoplasms, cerebral vascular disease, nasal sinus disease and the epilepsies.

In conclusion, symptomatic departures from classical migraine exist but frequently are not recognized as part of the migraine syndrome. These variants include transient phenomena referable to other systems than those classically associated with migraine. These include aura referable to various systems and organs, paresthesiae, aphasia, alexia, agraphia, vertigo, cerebellar dysfunction, paralysis of cranial nerves, neuralgia, monoplegia, hemiplegia, visceral or abdominal angina and fugue states. Many of these manifestations emphasize the concept and hypothesis that vasoconstriction and/or dilation underlie these transient functional interferences with nervous system activity. Many of the features of these types of cases come out only if specifically inquired about in obtaining the history in cases of recurrent headache.

REFERENCES

1. Moersch, F. P.: Psychic Manifestations in Migraine. Am. J. Psych., **3**:697, 1924.
2. Ely, F. A.: Migraine—Epilepsy Syndrome. Arch. Neur. and Psych., **24**:943, 1930.
3. Folly and Debenedetti: Migraine Ophthalmique Accompagnee d'Epilepsie. Rev. Neurol., **2**:635, 1931.
4. Hoch, P.: Über seltene Formen der Migräne. Arch. f. Psych., **97**:553-568, 1931.
5. Riley, H. A.: Migraine. Bulletin Neurol. Inst. N. Y., **2**:429, 1932.
6. Bassoe, P.: Migraine, J.A.M.A., **101**:599, 1933.
7. Riley, H. A., Brickner, R. M., and Soltz, S. E.: Unusual Types of Headache. Bulletin Neurol. Inst. N. Y., **4**:403-421, 1935-36.
8. Schumacher, G. A., and Wolff, H. G.: Experimental Studies in Headache. Arch. Neur. & Psych., **XIV**: 199, 1941.
9. Wilson, S. A. K.: Neurology. Williams and Wilkins, Baltimore, 1941.
10. Ford, F. R.: Diseases of Nervous System in Infancy and Childhood and Adolescence. Thomas, Springfield, Ill., 1944.
11. Engel, S. L., Ferris, E. B., Jr., and Romano, J.: Focal Encephalographic Changes During Scotomas of Migraine. Am. J. M. Science, **CCIX**: 650, 1945.
12. Nielsen, J. M.: Textbook of Clinical Neurology. Hoeber, N. Y., 1951.

New Books.

The Tompkins-McCaw Library of the Medical College of Virginia announces the following new books which may be had by our readers under usual library rules:

Advances in pediatrics. Vol. 5, 1952.
Barron—Modern trends in physiology and biochemistry. 1952.
Davidoff and Epstein—Abnormal pneumoencephalogram. 1950.
Duncan—Changes in the adrenal ascorbic acid and cholesterol levels following acute alcoholic intoxication. 1952.
Hahn—Manual of artificial radioisotope therapy. 1951.
Haas—Practical and occupational therapy for the mental and nervously ill. 2nd ed., 1946.
Henry—Extensile exposure applied to limb surgery. 1950.
Latimer—Utilization of the tonic neck and labyrinthine reflexes for the facilitation of work output. 1952.
Penick and Company—Bacitracin. 1952.
Pottenger—The fight against tuberculosis. 1952.
Recent progress in hormone research. Vol. 7. 1952.
Riseman—P Q R S T. 3rd ed., 1952.
Spitzer—Architecture of normal and malformed hearts. 1951.
Talbot—Functional endocrinology from birth through adolescence. 1952.
U. S. Atomic Energy Commission—Isotopes. A five year summary of U. S. distribution. 1951.
Willard and Spackman—Occupational therapy. 1947.

STEROID HORMONES AND RHEUMATISM
Report of Two Cases of Rheumatoid Arthritis

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A significant and probably causative relationship between alterations in adrenal cortical activity and rheumatoid arthritis has long been contemplated by the present authors and has been strongly borne out by their recent therapeutic experiences as well as by others. The following two cases are therefore reported in this connection, the first having metastatic involvement of the adrenals (proven at autopsy) with rheumatoid arthritis and encephalomalacia, and the second having mumps and "acute infectious arthritis", which, however, we believe to have been based on gonadal involvement and hepatic dysfunction, with associated abnormalities of steroid hormone metabolism.

CASE REPORTS.

Case 1. H. C., a 59 year old, white male was admitted January 15, 1947. When he first entered the hospital, he was found to be dazed and incoherent. It was also noted that his ankles and fingers were swollen.

History: Two years before he had developed chronic bilateral subacromial bursitis. He was treated for long periods with physiotherapy and traction. During this time he was admitted to hospitals twice and was discharged with a diagnosis of chronic *subdeltoid bursitis; arteriosclerotic vascular disease, generalized osteoarthritis, paraxysmal auricular flutter.*

In 1946 the patient had an episode of pneumonia followed by spontaneous right pneumothorax. He recovered after two months of hospital treatment. During the past year he has had periods of memory loss and of confusion, disorientation and hallucinations. These have increased in severity.

Physical Examination: The patient was undernourished. He appeared chronically ill. Temperature was 98.6; pulse 60; respiration 24; blood pressure was 160 systolic and 90 diastolic; weight was 155 pounds. The lungs were normal to auscultation and percussion. The heart was not enlarged to percussion and no murmurs were heard. There was a slow sinus arrhythmia. The abdomen was soft; liver, spleen and kidney were not palpable; no masses were felt. The prostate gland was normal. Reflexes were normal. There was marked limitation of motion of both shoulders, any movement producing severe pain.

Laboratory Data: The urine was negative on routine analysis. On January 16, 1947, the 17-ketosteroid excretion in the urine was 12.4 mg. in 24 hours, and on March 17, 1947, it was 2.8 mg. in 24 hours. The red blood count was 4,700,000. The hemoglobin was 87% of normal. The white blood count was 14,500. There were 80 per cent neutrophiles, 15 per cent lymphocytes, and five per cent eosinophiles. On March 20, 1947, the white blood count was 24,000 with 80 per cent neutrophiles, nine per cent lymphocytes, 11 per cent monocytes. Erythrocyte sedimentation rate was 80 mm. per hour and 87 mm. per hour Westergren method. The Wassermann test was negative. Sputum was negative for tubercle bacilli. Sputum cell block failed to show cells resembling carcinoma. Chemical examination of the blood disclosed: Glucose 98 mg. per cent; urea nitrogen 26.8 mg. per cent; serum cholesterol 197 mg. per cent; cholesterol esters 110 mg. per cent; total proteins 6.0 gm. with albumin 3.6 gm. and globulin 2.4 gm. per 100 cc. Examination of the spinal fluid revealed a protein concentration of 69 mg. per cent, and there were four lymphocytes per cc.

The electrocardiogram showed left axis deviation and auricular extra-systoles.

Roentgen-ray examination of the chest on admission showed an area of infiltration a half inch in diameter in left upper lobe. (Plate I) Further x-rays of chests taken between January 16 and March 27 continued to show this area. It was suggested that it might be neoplastic in character. X-ray of the skull was normal. Pneumoencephalograms revealed excessive air over the surface of the cerebral hemispheres. A diagnosis of cerebral atrophy was

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made. Calcium deposits were noted in both subdeltoid bursae in x-ray films taken of the shoulder. Hand plates showed changes typical of rheumatoid arthritis.

Course: During January the patient continued to be sluggish, semi-comatose, irrational and confused, although at times he was well oriented. In February this condition of episodic disorientation continued. He complained of pain in his arms, legs, shoulders and hands, and most of his other joints.

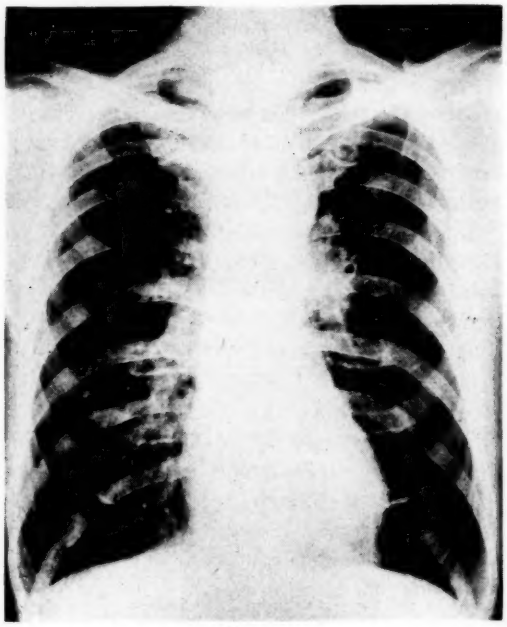

Plate I.

By the end of February these pains had decreased, and there was less pain than before on moving about. His therapy during these months had been testosterone and monomestrol daily, and demerol for pain. During the first week of March he developed pain in his left shoulder and leg. He was given three infusions of 250 cc. of 0.1 per cent procaine intravenously on alternate days. During the second week of March he complained of pain in his right leg and arm. He could, however, move his left leg more freely and later had less pain in right leg and left arm. By the middle of March he was able to get out of bed in a wheel-chair. His mental confusion continued unchanged. Later in the month the generalized pain returned. The testosterone and monomestrol administration was stopped. On March 27 his condition become very poor, and broncho-pneumonia developed. His temperature was 102 degrees, his pulse 144. He was given penicillin, but his course continued downhill and he expired March 29.

Necropsy: There was general malnutrition. There was fusiform swelling of interphalangeal joints of both hands, and the knee joints were fixed in 40 degree flexion. The mediastinal lymph nodes were enlarged. The right lung weighed 750 gm.; left lung weighed 550 gm.; and the secondary bronchii of the latter showed tumor tissue. The liver weighed 2,500 gm. and was congested; spleen weighed 325 gm. and was grossly normal. The right adrenal weighed 75 gm., measured 7 by 2.5 cm., and a discrete white tumor nodule, 4 by 2.5 by 5 cm. replaced most of the gland; the left adrenal weighed 175 gm., measured 8 by 7 by 3 cm., and was entirely replaced by tumor tissue. The brain was normal. Microscopic anatomy; the right lung showed broncho-pneumonia of the right lower lobe and healed tuberculosis in the apex. There was metastatic bronchial carcinoma of the adrenals (Plate II).

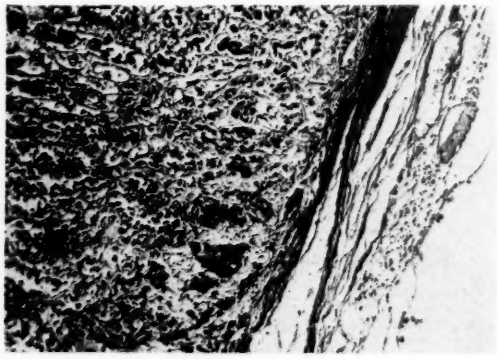

Plate II.

Anatomical diagnosis: Bronchogenic carcinoma, left upper lobe, with metastases to mediastinal and para-aortic lymph nodes and to both adrenal glands. Broncho-pneumonia, right lower lobe. Healed tuberculosis, apex of right lung. Rheumatoid arthritis.

This case is presented not because it is proposed that there is a direct association between carcinoma of the lung and rheumatoid arthritis, but to show the relationship of change in activity of the adrenal cortex to the cause and course of rheumatoid arthritis. There may be, however, some relation between carcinoma of the lung and pulmonary osteoarthropathy. Sometimes cases of carcinoma of the lung present changes that point to a diagnosis of rheumatoid arthritis.[1] This was noted in the case of a male patient

in whom, at post-mortem, there was discovered an adenocarcinoma of the lung with metastases to the adrenal cortex. The 17-ketosteroid excretion had been 8.2 mg. in 24 hours.[2]

Case 2. The classification of arthritis occasionally includes, under the terminology of arthritis due to specific cause, the arthritis of mumps. This has been called a virus arthritis, and such a case is presented below.

A search of the literature reveals only one comprehensive article on this subject. This was published as a thesis in France by Maisondieu in 1924.[3] In the 2668 cases of mumps reviewed, he found arthritis in 0.44 per cent. We feel that the case so reported indicates that perhaps the severe rheumatoid arthritis resulted from the testicular involvement and not from a virus directly. There is also the possibility that hepatic involvement might so interfere with steroid metabolism as to have produced the arthritis.

The patient, J.D., age 34, a married physician with two children, was admitted on January 20, 1935, complaining of pain and swelling in both parotid regions, of one day's duration.

His past history contained two significant features: (1) At the age of 14 he had been confined to bed for 2 or 3 days with pain and swelling in left great toe. (2) He had had from time to time a rather marked bradycardia (rate 40 to 50) following a blow over the precordium. On occasions there was electrocardiographic evidence of a sino-auricular block. He had, however, been unusually healthy and very active.

His admission pulse was 56, blood pressure was 126/72. The leucocyte count was 7,550. There were 61% polymorphonuclears.

On January 22, his left testicle became painful. On January 23, he became nauseated and momentarily lost consciousness; he complained of severe headache and intense nausea. During the night that followed, his temperature by mouth reached 102.4 degrees. On January 24, nausea and headache persisted, and he began to vomit. He could retain only orange juice, Vichy water, and small amounts of champagne. There was some stiffness of the neck. There were abdominal pain and tenderness. The spleen was thought to be palpable. The parotids were less swollen. The pulse varied between 80 and 100, and the blood pressure was 134/90. The rhythm was regular and there were no murmurs. The electrocardiogram showed markedly prolonged PR conduction time (0.4 sec.).

On January 26, the patient again fainted following an enema. After this, carpopedal spasm was present in both hands. The left hand remained in the "main accoucheur" position for several minutes. The Chvostek sign was present and very active. His blood serum calcium at this time was 8.7 mgs. per 100 cc. The blood pressure was 144/80. He was given glucose, saline and calcium gluconate intravenously. This tetany was thought to be due to alkalosis induced by the low chloride consumption and the high alkaline salt intake.

On January 27 the left testicle became enlarged and very tender, while the right testicle had been asymptomatic after the second day of the disease. There was also severe pain in the right side of the abdomen and in the epigastrium. The abdomen was rigid, especially on the right side. That evening the patient complained of numbness in the right hand and was apprehensive of another attack of tetany. Forty-five minutes later he developed a chill which lasted about fifteen minutes. He became pale and cyanotic. The Chvostek sign was not so active as on the day before. His serum calcium was 8.8 mgs. per 100 cc. and the phosphorus was 4.1 mgms. per 100 cc. The temperature rose to 104.4 degrees.

By January 28, the swelling in the parotid glands had almost disappeared and the submaxillary glands were no longer palpable. There was now no stiffness of the neck. The pain in the epigastrium was still present. His blood pressure was 150/80.

On January 29, following codeine administration, his respirations fell to 11 per minute. It was thought that there was involvement of the central nervous system.

By now the parotid swelling had entirely subsided but the opening of the left Stenson's duct was still red.

On February 1, his temperature was normal and he began to feel well again. His pulse remained between 55 and 60 per minute.

On February 3, he complained of soreness deep in his throat and of an irritating rash on the knees and arms. The rash at first had the appearance of small petechial areas. A throat culture showed alpha hemolytic streptococci. No beta organisms were found.

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About noon on this day, the 14th day of the disease, he began to complain of pain in his shoulders and back. This became steadily more acute and by 3 P.M. it was described as unbearable. The maximum intensity was in both shoulders. The arms could not be raised above 20 degrees. The muscles of the neck and back were also involved. The weight of the bed clothes or the pressure of the bed on his back was excruciatingly painful. There was exquisite tenderness over both acromial processes, with doubtful or slight swelling in the region of tenderness. He was given aspirin, 15 gr., and codein, 1 gr.

On February 4, there was swelling over both shoulder joints in the region of the acromio-coracoid bursa. There was no redness or heat. Any motion was acutely painful. The rash was still present on the inner sides of the knees and elbows. The blood proteins were low and there was a reversal of the albumin-globulin ration. The white count was 13,000 with polymorphonuclears 63%. The sedimentation rate was 31 mm., the temperature 98. By February 5, the testicular discomfort and swelling had entirely gone, and the pain in the shoulders was less, but motion of the fingers of the left hand began to be painful. Later in the day, both hands became involved and there was typical fusiform swelling of the finger joints. The rash, which was follicular in distribution, began to spread. It had the appearance of a dyschotic eczema. It was suggested that the patient did not have rheumatic fever and that the rash was a folliculitis. It is interesting that the type and appearance of the rash varied somewhat from day to day.

By February 10, he seemed much better but the rash was more extensive. The possibility of a bromide rash was considered, but there was little evidence for this. It was thought by some that the rash was a toxic dermatitis.

On February 13, the right wrist and right knee became quite painful. There was no redness, tenderness or heat. The right shoulder was still slightly painful, but the pain disappeared by the next day and he was allowed out of bed. Sedimentation rate was 11 and blood pressure had returned to 126/78. He was discharged on February 18, with the following diagnosis: (1) Mumps (2) Acute pancreatitis (3) Acute infectious arthritis (4) Acute orchitis (5) Eczema (6) Cardiac diagnosis unknown. Encephalitis was not proven. He did not regain complete use of his right shoulder until four weeks later. At the present writing, fourteen years later, he is entirely well. There has been no return of the arthritis. The electrocardiogram is normal, the blood pressure is 128/84 and the pulse is 76.

The severe nausea and vomiting which preceded the attack of acute arthritis, the very foul stools, and the abdominal pain may have been due to hepatitis as well as pancreatitis. There is no history of jaundice in this case, although it is fairly common in patients with mumps. The low total protein and the abnormal albumin-globulin ratio are suggestive of liver damage.

It is not thought that he had rheumatic fever for the following reasons: (1) Alpha hemolytic streptococci only were found on culture from mild pharyngitis. (2) Fusiform swelling of fingers was present. (3) There was a poor response to salicylates. (4) There were no typical EKG changes, other than the prolonged PR interval which was present before the attack. (5) No subsequent attacks occurred in later years. (6) Evidence of hepatic disturbance was present. (7) Involvement of shoulders was predominant. (8) Sedimentation rate returned suddenly to normal. (9) Recovery was rapid. (10) At the time of the arthritis there was no elevation of temperature. (11) There was no evidence of anemia. (12) The arthritis was severe. (13) There were no cardiac murmurs.

It is to be regretted that we were unable to determine 17-ketosteroids on this patient. In the presence of the severe arthritis, both the 17-keto and the 11-oxy steroids levels were most probably changed. The hepatitis, too, may have contributed to this dysfunction.

These two cases are reported because it is felt that severe arthritis in the first instance is associated with involvement of the adrenal cortex, while, in the second, it may be a result of gonadal involvement and hepatic dysfunction.

BIBLIOGRAPHY

1. Annotations: Joint Changes in Cancer of the Lung, Lancet, **252**:378, 1947.
2. Case Records, Mass. Gen. Hosp.: Case 31281, New Eng. Med. J., **233**:44, 1945.
3. Maisondieu, Prosper: Etude des Manifestations Articulaires des Creilloms, Thesis for M.D., Faculte de Medicine de Paris, 1924.

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ACUTE PORPHYRIA AND ONE FALSE INITIAL DIAGNOSIS DUE TO THE PREVIOUS TAKING OF PYRIDIUM*†

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Porphyria is not a common disease. That it occurs more frequently than it is diagnosed generally is indicated by the number of relatives of some patients in whom there has been chemical evidence of the abnormality associated either with mild symptoms or with none at all.

Its recognition is important because it may imitate almost any abdominal or neuropsychiatric disease. Because of it, many patients have had useless operations. One of our four patients had had an appendectomy. Another previously had been diagnosed acute pancreatitis. The diagnosis usually is easily made by having the condition in mind, observing the red to mahogany-colored urine when it is fresh or after it has stood, and the performance of a simple chemical test, the significance of which will be discussed.

Porphyria is the result of an inborn error of metabolism. That this is true, even of the more common acute type to which our patients belong and which usually becomes clinically manifest in middle adult life, is indicated by the familial occurrence. Such patients may previously have noted an unusually dark or red urine. They may also previously have had episodes of abdominal or neuropsychiatric disturbances, as did two of our patients.

The chemistry of the porphyrins is a complicated subject. Our knowledge concerning it is not complete, despite the considerable progress that has been made, in recent years, in this country and in England[1,2,3]. A few simple facts will suffice for our purpose.

The porphyrins are the basis of the respiratory enzymes which occur in all living cells. Their chemical basis is four pyrrole groups linked together by carbon atoms to form a ring and there are various side chains. When iron enters the center of the ring and the group combines with different specific proteins, hemoglobin, myoglobin, catalase, peroxidase and cytochromes are formed.

There is evidence that coproporphyrin is the only porphyrin normally occurring in urine. An increased excretion of coproporphyrin occurs in a large number of diseases and poisonings. That excretion, in itself, is not known to be harmful. The condition is named porphyrinuria, in contrast to porphyria with which we are concerned.

The porphyrias have an increased excretion, in urine and feces, of abnormal amounts of both uroporphyrin and coproporphyrin. The determination of the more specific uroporphyrin is not feasible in most laboratories. However, cases of acute porphyria with abdominal or neurological manifestations have been found by Watson[4] regularly to excrete in the urine a third substance known as porphobilinogen. This substance has not been crystallized and its chemical relationships have not been determined, but it is easily recognized by the test described by Watson and Schwartz[5]. That is the development of a red color with Ehrlich's reagent which is not extracted by chloroform as is the red substance which Ehrlich's reagent forms with urobilinogen.

There are three types of porphyrias. The congenital type is rare. In it, photosensitivity is predominant and the skin lesions may be serious. Some have had splenomegaly and hemolytic anemias. Splenectomy has relieved not only the anemia but also the photosensitivity[3]. Porphobilinogen has been absent from the urine[4].

The "mixed" type, beginning later in life, is also uncommon. It occurs mostly in alcoholics[6]. It is relatively benign, also with photosensitivity and the manifestations chiefly dermatological. The chemical findings are variable, but porphobilinogen was

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found by Watson[4] only in those having abdominal or neurologic manifestations.

Acute porphyria is really a chronic disease with acute exacerbations. In the past, a large minority of episodes were attributed to certain drugs, particularly sulfonal, trional and barbital. Drugs have not been frequently incriminated in the more recently reported cases.

No single pattern can be described for the attacks. The abdominal pain has commonly been described as cramp-like. It may be intermittent but continuous and severe when present. It may be generalized or localized to any part of the abdomen or flanks. Muscle spasm has been described in a few cases, but usually there is a lack of spasm or rebound tenderness. On the other hand, patients may have fever and leukocytosis suggesting inflammatory disease. Marked constipation is usual, but diarrhea has been described. Abdominal distention may occur. X-rays may show dilatation of the stomach or of segments of bowel, but seldom, if ever, show the characteristic pattern of intestinal obstruction. Jaundice and impairment of liver function occur in some cases. Hypertension is found in some cases and it may be intermittent, even with periods of hypotension.

The nervous manifestations are likewise extremely varied. Before the acute episode the patient may have had lesser neurospychiatric symptoms for years. Severe pain in the extremities may precede weakness or paralysis. Paralysis may develop suddenly or over the course of weeks. The distribution is usually irregular but in the severe cases becomes almost universal. The mental picture may range from normal through apathy and confusion to a toxic psychosis or coma. Death is usually from respiratory failure.

The diagnosis in either the abdominal or the neurological type, there being no characteristic clinical picture, depends upon considering the condition as a possibility and testing it in the laboratory. The urine may be red or mahogany-colored when it is passed or, if the specimen is acid, it may become so on standing, particularly if exposed to sunlight. It is well to have the laboratory alerted to call attention to all unusually colored urines. The word "dark" on a report may not impress the clinician.

The Watson-Schwartz test[5] for porphobilinogen is easily performed and is usually reliable. The test may become negative if the urine has stood and should be done on relatively fresh specimens. Watson found[4] that the urines of forty-four patients with acute porphyria in relapse all contained porphobilinogen. This chromogen diminished or disappeared during remissions. It was not found in the urine of four patients with the congenital photosensitive type of porphyria. In eight patients with the "mixed" type it was found only in the urines of those having abdominal pain or neurologic disturbances.

Watson has stated[7] that several instances of a false red or pink color in the porphobilinogen test have occurred in his laboratory. One was a case of acute poliomyelitis with large amounts of coproporphyrin and urobilinogen in the urine. Another was a cirrhotic whose urine contained large amounts of coproporphyrin. With several urine specimens containing large amounts of urobilinogen, not all of the red reaction compound could be extracted by chloroform. Crayons and beets ingested by infants gave weak but definitely positive porphobilinogen reactions. In another instance the red color, disappearing like an indicator on alkalinization, was thought to be due to an extrinsic pigment. Hammon and Welcker[8] found no positive tests in examining the urines of 1000 hospital patients.

In our last case who had a peripheral neuritis, the finding of a red urine led to an original false impression of porphyria. The suspicion seemed at first to be confirmed by the Watson-Schwartz test, although the color found in it was rather more orange than the bluish red color of the reaction with porphobilinogen. It was subsequently learned that the patient had been taking pyridium. Other patients were given pyridium and their urines then gave similar false positive tests.

The mortality in the nervous type is high—over 50% in the first attack. The prognosis in the abdominal type is much better.

There have been some reports of improvement following the use of liver extract or other vitamins, but it has generally been found that no treatment is specific. Management remains, at present, symptomatic and supportive. Demerol, because of its antispasmodic effect, has been more effective than morphine for abdominal pain, although of shorter effect. It seems wise to avoid barbiturates because of the evidence that they have participated acute episodes.

CASE REPORTS

Case 1: A white man, twenty-five years old, was

referred to the hospital on March 26, 1951, with a diagnosis of acute pancreatitis. He complained of a recurring, severe paraumbilical abdominal pain of five days' duration. Three years and one year prior to admission he had experienced similar attacks. In the 2nd episode he was diagnosed pancreatitis in another hospital. Physical examination revealed a well-developed and well-nourished man, complaining bitterly of abdominal pain. He had a soft abdomen, no fever, and a blood pressure of 160/120. The urine was dark reddish in color and gave a positive Watson-Schwartz test for porphobilinogen. A 24-hour specimen of urine contained 1240 micrograms of coproporphyrin and was positive for uroporphyrin. Routine laboratory reports and the serum amylase content were reported as normal. The pain was of such severity as to require methadon, 7.5 mgms. subcutaneously, q. 4 h. plus demerol, 150 mgms. subcutaneously, q. 1 to 2 h. for approximately 10 days following admission. From this time on improvement was gradual, and on April 21, 1951, he was free of pain and his blood pressure was normal. He remained asyptomatic and was discharged April 26, 1951. Treatment was symptomatic.

Case 2: A white man, twenty-three years old, entered the hospital in March, 1950, complaining of lower abdominal pain, nausea, and generalized muscular weakness of three weeks' duration. An appendectomy had been performed at another hospital on account of the abdominal pain, but the operation afforded no relief. Physical examination revealed marked generalized weakness, amounting almost to paralysis of many muscles, hypoactive deep tendon reflexes, soft abdomen, blood pressure 160/120, and no fever. The routine laboratory reports were normal. The urine gave a positive reaction for porphobilinogen by the Watson-Schwartz method, and was also positive for uroporphyrin and coproporphyrin. The patient remained afebrile. Treatment was symptomatic. Improvement was gradual. Six weeks after admission his blood pressure was normal, he was asymptomatic, and there were no findings suggestive of neurological disease.

Case 3: A white man, fifty years old, entered the hospital on November 20, 1947, because of hypertension of many years' duration. Twelve days after admission he began having severe mid-epigastric pain, nausea and vomiting. The abdomen was soft, he had no fever, and was constipated. The routine laboratory reports, gastro-intestinal x-ray series and barium enema were normal. On December 20, 1947, porphyria was considered as a possibility, and his urine at that time showed the presence of porphyrins by the ether extraction method. Later, urinary porphobilinogen was demonstrated, and on February 20, 1948, a 24-hour specimen of urine was reported as containing 1500 micrograms of uroporphyrin. Treatment was symptomatic and recovery was uneventful.

Case 4: A Negro man, forty years old, was admitted to the hospital on September 8, 1949, complaining of abdominal pain, severe constipation, nausea and vomiting, numbness, tingling and weakness of the lower extremities. During the 12 months prior to admission he had experienced three similar attacks, all occurring after he had been on a spree of from 1 to 2 weeks' duration. The symptoms did not occur as long as he was on the spree, but developed within 24 to 48 hours after cessation of alcoholic consumption. With each episode he noted that his urine was dark red and his feces were darker than normal. The physical examination revealed a well-developed, slightly dehydrated Negro man, with a tender, moderately spastic abdomen, temperature of 100.8° F., blood pressure 170/114, muscular weakness of the lower extremities with hypoactive deep tendon reflexes. The routine laboratory reports were normal. One specimen of urine was reported as giving a negative test for porphobilinogen by the Watson-Schwartz method. He was treated symptomatically, and 3 days after admission showed marked improvement objectively and subjectively. At that time it was necessary for him to leave the hospital to attend to some business, and he did not return. We are aware that this is not a proven case of porphyria, but include it because of the clinical history, the neurological findings, and his red urine. We are not sure that the porphobilinogen test was done on a fresh specimen. Tests for uroporphyrin were not properly done.

Case 5: A white man, twenty-seven years old, was admitted to the hospital on April 15, 1951, complaining of abdominal pain, generalized muscular weakness, and numbness and tingling of his feet of one week's duration. The physical examination showed muscular weakness of the upper and lower extremities with hypoactive deep tendon reflexes, partial right facial paralysis, soft, tender ab-

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domen, temperature 100° F., and blood pressure normal. His urine was reddish and was reported as positive for porphobilinogen by the Watson-Schwartz method. The spinal fluid contained 370 mgms.% total protein and 20 w.b.c. The routine laboratory reports were normal. Gastro-intestinal x-ray, gall bladder series and barium enema were reported as normal. Subsequent Watson-Schwartz tests were reported as negative. It was learned from the patient's local physician that for several days prior to admission the patient had been taking pyridium. This drug was given to several patients on the Ward. Their urines then gave similar false positive tests for porphobilinogen. The patient thus had the findings of the Guillaine-Barre syndrome. His red urine and the test for porphobilinogen at the time of his admission, together with his clinical picture, led to an original false impression of acute porphyria.

SUMMARY

Four cases of acute porphyria are presented. Another patient having peripheral neuritis was originally so misdiagnosed because of red urine, giving a false positive test for porphobilinogen, due to pyridium. Acute porphyria, although not common, occurs frequently enough so that its possibility should be considered in any abdominal or neuropsychiatric disorder which is not otherwise clearly explained.

BIBLIOGRAPHY

1. Welcker, M. L.: The Porphyrins. N.E.J.Med. **232**:11, Jan. 4, 1945.
2. McSwiney, R. R., Nicholas, R. E. H., and Prunty, F. T. G.: The Porphyrins of Acute Porphyria. The Detection of Hitherto Unrecognized Porphyrins. Biochem. J. **46**:147, Feb., 1950.
3. Watson, C. J.: Some Recent Studies of Porphyrin Metabolism and Porphyria. Lancet **1**:539, March 10, 1951.
4. Watson, C. J.: Urinary Findings in Relation to the Manifestations of Porphyria. J. Lab. & Clin. Med. **36**:1002, Dec., 1950.
5. Watson, C. J., and Schwartz, S.: Simple Test for Urinary Porphobilinogen. Proc. Soc. Exp. Biol. & Med. **47**:393, June, 1941.
6. Brunsting, L. A., Mason, H. L., and Aldrich, R. A.: Adult Form of Chronic Porphyria with Cutaneous Manifestations. J.A.M.A. **146**:1207, July 28, 1951.
7. Watson, C. J.: Quoted by Hammond and Welcker[8].
8. Hammond, R. L., and Welcker, M. L.: Porphobilinogen Tests on a Thousand Miscellaneous Patients in a Search for False Positive Reactions. J. J. Lab. & Clin. Med. **33**:1254, Oct., 1948.

Magnetic Sound-On-Film Will Aid Medical Teaching.

The addition of a magnetic iron stripe to 16mm film will produce a small revolution in all education, according to Dr. David S. Ruhe, Director of the Medical Audio-Visual Institute of the Association of American Medical Colleges.

The magnetic stripe is a recent technical development which allows amateur film producers to convert silent film footage into sound movies easily and inexpensively.

Writing in the May 1952 issue of *The Journal of Medical Education*, Dr. Ruhe goes on to say that in medical education this revolution is coming very soon. Rapid advances of medical research projects make constant editing of teaching films necessary in order to prevent obsolescence. This problem now becomes greatly simplified.

Making use of the same procedure as the tape recorder, the stripe makes possible great flexibility in commentary. Medical teachers will be able to adapt sound strips to different audiences. For example, a film used for medical students may be "erased" and re-recorded for use with a group of nurses.

Cost of the magnetic iron stripe will be somewhat less than the titles of a silent film, Dr. Ruhe says.

Many medical centers possess silent films of surgical procedures and rare clinical cases which may now be adapted easily for classroom use. Projection time is gained with the omission of titles.

Quality of sound on the magnetic film is excellent, and the iron stripe is said to outwear the film to which it is applied.

HOW YOUNG AM I?*

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The time that old age begins is debatable. At the present time, it is largely a matter of personal opinion or definition. Most people like to believe that old age begins about ten years beyond their current age. The teenager plans to "be old" at 25. The matron at 55 is resigned to the fact that at 65 she will have to accept the classification of "oldster."

A comprehensive survey made to obtain a more satisfactory answer to this question showed that a majority of the people questioned felt that 49 marks the entrance to old age.

Did any of you see that recent Robert L. Ripley cartoon, captioned: "The Only Women in the World Who Never Grow Old?" It depicted the women members of a tribe in India who were born 60 years old. Each year following birth, they subtracted one year from their age. They kept getting younger and younger, instead of older and older. That's a good trick, if you can do it!

Aging is the result of a very complex interaction of biological forces, most of which are unpredictable and cannot be measured with any degree of accuracy.

Aging should not be confused with longevity. Longevity merely is a measure of the number of years we live. As far as oldsters go, there isn't much that we can do about that.

All kinds of people with all kinds of habits, both good and bad, manage to become centenarians. Some eat, drink, and smoke to excess, others just loaf along through the years. The only common denominator found in the past histories of most centenarians is the fact that their parents lived a long time. Their longevity seems to have been inherited.

Perhaps that's the very clue we have been looking for. If we want to live to be 100, and if we ever again have the opportunity, let's be sure to select long-lived parents.

We still know very little about the aging process, but we do know a lot more about it today than we did fifty years ago. It is now believed that various diseases and unfavorable environmental conditions join hands to bring about a cumulative breakdown of the organs and tissues of our bodies. This breakdown is spotty. Some tissues age faster than others. As tissues are destroyed, little by little they are replaced by a form of less efficient connective tissue. You have seen the evidence of that in the wrinkling of the skin on the back of the hands with age. When the breaking down process reaches its maximum, we die.

It is the long period of gradual breakdown prior to death which we call the period of "aging." This period may be short or it may be very long, depending upon a lot of variable factors which are difficult to define. For example, a person whose heart ages much faster than other parts of the body may die at the age of 50, of coronary heart disease, whereas another person whose organs all age at about the same rate may live to be 80. If only some way could be found to minimize the injurious effects of disease and environment, life probably could be prolonged indefinitely.

As Dr. Henry Simms, of the College of Physicians and Surgeons, in New York City, puts it, "It has been computed that if the human body could retain all during life the ability to resist disease and repair breakdown that it possesses at the age of 10, man would have a life expectancy of 800 years and some individuals might survive 2200 years." Eight hundred years is a long time—perhaps too long.

Aging actually starts at the time of conception, but it only becomes apparent as "aging" in the later years of life. From conception on, the various organs and tissues go through a period of involution that eventually terminates in death.

For a few years after birth, the tissues grow faster than they break down. That is the period of childhood, when maximum growth occurs. Sooner or later, the reparative process begins to lose out. It is then that we begin to notice that the stairs have grown steeper and longer, that lights are less bright; and that our daily workload *must* be heavier because we are much more tired at night than we used to be. This is evidence that age is creeping up on us.

This process of breaking down is not related very

*Read before the Health Meeting of the Business and Professional Women's Club of Richmond, Va.

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closely to the actual number of years we have lived. It is much *more* related to our biological age—not our chronological age.

Most of us know what our chronological age is: 32, or 45, or 57. None of us knows our biological age. At any given time, our brain, biologically may be 50, our heart, 70, and our liver, 100. The only way we, theoretically, could determine our true biological age would be to determine the separate biological ages of each of our organs and tissues and then average them out. We can't do that with the means at hand today. The best we can do is to estimate our biological age by indirect methods.

We *can* have a physical examination. If this is done by a competent and interested physician, who makes full use of the many laboratory tests that are available, we can establish a close approximation of our biological age. Such an examination cannot be considered complete unless the mental and emotional status is determined at the same time that the physical examination is done.

Many early chronic diseases, which otherwise would cause premature aging, are picked up during these periodic examinations. Found early, many can be brought under control and undue aging can be prevented.

The very interesting and successful multiple screening demonstration which was conducted in Richmond, Virginia, last year, was indicative of the growing tendency to seek out signs of the chronic diseases early, so that they can be stabilized.

It is hard to appreciate the rapid increase in the incidence of chronic diseases during the last two decades. Recently I received a copy of the monthly bulletin published by the Virginia State Health Department. In it I found a table that listed the number and causes of deaths in Virginia for the first six months of 1950. Of the 14,755 deaths recorded, all except 4,896 of the deaths were caused by the cardiovascular diseases, cancer, and accidents. Two-thirds of all the deaths in Virginia for the first six months of this year were attributable to these three causes.

As the number of older people in our population continues to skyrocket, because of better medical and public health practices, the incidence of the chronic diseases will continue to increase. This is inevitable.

The control of the chronic diseases bears a definite relationship to aging. The chronic diseases accelerate the process of physical deterioration and retard the normal processes of repair. They result in premature aging and many times, premature death.

At times, the human race is very inconsistent, some times more than others. There is one point of inconsistency which, to a physician, stands out above all of the rest. Billboard advertising, radio and television exhortation, and the soft-spoken suggestions of service station attendants have succeeded in conditioning most of us to husband the mechanical resources of our automobiles, not that there's anything wrong with that. It's a commendable practice. The regrettable fact is that physicians, public health workers, and health educators, including myself, somehow have failed, where the automotive industry has succeeded. We have never succeeded in conditioning large numbers of people to take stock of their physical, emotional, and mental equipment in the same manner and to the same degree they have their cars checked.

The wisest thing that we can do is to admit our failure gracefully, borrow some lessons from our commercial brethren, if that will help, and develop more realistic and effective methods of getting the idea over to people that they need to do something in a positive way to preserve what health they have left.

When, some years back, I became interested in aging as a public health problem, I, naturally enough, was curious to find out why aging and the control of chronic illnesses had suddenly become a national problem. The facts which confronted me soon convinced me that this was a huge problem.

In 1900 there were only three million people in the United States over the age of sixty-five. By 1950 this number had risen to eleven and one-half million. It is estimated by 1980 the number will be about twenty-two million. That's a big problem. We are being engulfed by a veritable flood of oldsters.

The economic importance of the problem was as impressive as its size. Three-fourths of the eleven and one-half million oldsters receive an annual income of less than $1000 a year, from all sources, which is about $80 a month. There *was* a time when you could stretch a thousand dollars a long way. An income of $1000 a year today merely provides one with a delightful opportunity to slowly starve to death.

In his summation of the proceedings of the 1950

Conference on Aging, Dwight Cook reported that, ". . . if present trends continue, by 1957 the support of the aged will cost the Federal treasury fifteen billion dollars a year." In New York State, the care of the aged represents the largest single public welfare expenditure in the State. On September 1st, a news release quoted the increase in the "over 65" age group in Washington, D. C.—from 1940 to 1950—as 15,610, or 37%. This was the highest increase noted in any age category. The day before, another news item stated that the increase in the size of the older age group was creating many difficulties in the administration of the public welfare program because so many people in this age group were candidates for one sort of relief or another.

It looks as if we will have to figure out some way to keep these older folks at work as long as possible if we are ever going to be able to handle this problem.

Back in 1880, when most people lived on farms, this was no problem. Seventy per cent of the men over sixty-five were working. The women were taking care of the house. In 1950, with the majority of the population residing in cities and towns, about forty-three per cent of the men over sixty-five are working. Estimates indicate that if the present downward trend continues until 1960, only *one* out of every *three* men over sixty-five will have a job. The statistics for women are equally disturbing. Only nine per cent of older women are employed. The full significance of these facts is not hard to appreciate.

By 1960, two out of every three men over sixty-five, and better than nine out of every ten women in that same age group will have to be supported by those of us who are still working. If the number of people over sixty-five in the population continues to mount, and the number of people over sixty-five who are working continues to drop, imagine the economic burden that middle aged workers will have to carry ten or twenty or thirty years from now.

If some method is not developed that will turn these non-producers into producers, the financial burden eventually will become back-breaking.

It would almost seem that medical and public health practices were working to unbalance our national economy. I have confidence that we can continue to save and prolong lives without wrecking our economy. All that has to be done is to take the necessary steps to correct the situation that has developed.

There are several things that we can do right now.

1. We can take time out from our otherwise busy lives to really understand the aging problem and all of its many ramifications.

2. We can join with others to change our archaic system of retirement. People are hired because they are fit to work. They should be fired or retired because they no longer are fit to work—not because they have reached some arbitrarily established chronological age.

3. We can encourage and assist in the development of rehabilitation and re-training services, so that periodically, older workers can be "de-aged." By physical and mental rehabilitation, we can salvage many discarded workers. Re-training older people for less arduous jobs can salvage many others. We need them all on the production lines.

In addition to its size and its economic importance, the problem of aging has great social significance. In the words of Dr. Martin Gumpert, an eminent geriatrician, "The change that has occurred almost imperceptibly implies a profound revolution, a revolution in deep-rooted standards of life, of thought, of social and economic conditions, of family bonds, which may crack or rebuild the foundations of our civilization."

For many years, we have been focusing our attention on children and youth. Much less thought has been given to the care of the aged. As a result of this social myopia, many old people are hungry; many lack adequate shelter; thousands die each year in institutions, abandoned by their families and friends; still others live unhappy, frustrated lives with sons, daughters, or relatives whose knowledge of how to deal with children far exceeds their appreciation of the basic needs of the old folks.

Many of these needs are intangible. Others are very tangible. There are five basic ones.

1. Old folks need adequate medical care if they are to be kept in a state of reasonable repair.

2. They need economic security—not an income of $5000 a year, but enough to guarantee them the essentials of life.

3. They need an opportunity to engage in useful activity. It has been well said, "A mind at rest is a mind distrest."

4. They need a home. A house is not enough.

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They need a home in which they can have some privacy, and a quiet place to read . . . a home in which they will be wanted and appreciated.

5. They need to maintain their independence and their self respect. It is tragic when these are taken away from them. It is going to take some knowledge of how the human mind works for us to understand and apply these five basic principles.

The whole problem of aging has been complicated and a solution to this problem has been delayed by the existence of faulty attitudes on the part of old folks themselves and on the part of persons responsible for their care. An example of this is the growing tendency for the care of aging parents to be shifted to already overloaded public agencies. Parents are declared to be mentally incompetent and are committed thoughtlessly to mental institutions.

82% of old folks committed to these institutions are admitted with a diagnosis of senile psychosis or cerebral arteriosclerosis. In layman's language this means that age has caused a hardening of the arteries of their brain. Their brain doesn't function as well as it did when they were in their prime. Most of these people are not dangerous, and treatment will be of little permanent benefit to them. They merely are being penalized for the crime of growing old and getting in younger people's way.

No matter how well constructed a mental hospital may be; no matter how good the food and no matter how skilled and considerate the staff, they are not the place in which most of us would want to end our days. Then why do so many children commit their parents to these institutions? Is it because they are selfish and cruel? Or is it because they are desperate and don't know where to turn for help in a community that, through ignorance of the problem, turns its back?

I don't think many of them mean to be selfish and cruel. With more community understanding and help most of these older folks could be kept at home.

* * *

There are other ways in which the rapid aging of our population is bringing about social changes. An example of this is our changing attitude toward the employment of older women. More women over 45 are employed in industry today than were employed during the year of peak war production—1944.

When you come right down to it, it makes sense to help these older women find employment. Once the children are raised, there is little reason why "ex-mothers" shouldn't seek employment, or re-employment, even if they obtain only part-time work. As important as bridge, canasta, and late afternoon cocktails are to our American way of life, they don't add much to the family income. At best, they are relatively benign escape mechanisms for a lot of bored people. They hardly qualify as a substitute for the very active and purposeful life that mothers live during the years that their children are growing up. On the other hand, sensibly selected employment permits ex-mothers to make a satisfying contribution to the welfare of their community and it gives purpose to their life which is all too often lacking.

Many strong influences are at work tending to favor the employment of older women. One of these is the growing need for increased production. Our standard of living is the result of an interplay between two factors: namely, the number of producers, and the number of consumers.

This can be visualized as a formula: $\frac{P}{C} = S$

The number of consumers is represented by all of us. Everyone is a consumer. The number of producers varies from year to year, dependent upon economic conditions, war, and politics. If the number of consumers remains substantially the same, whereas the number of producers increases, we will have more goods and services to distribute to the consumers. In other words, the factor "S", which represents our standard of living will go up. There will be more goods, such as refrigerators and nylons, and other public and private services—for all of us—regardless of cash income.

One way to increase the number of producers, and thereby raise our standard of living, is to encourage and make possible the employment of older women in positions fitted to their abilities, and geared to their biological age.

* * *

This business of growing old requires careful planning and preparation *before* we grow old—not afterwards. If we plan to work as long as possible, we may have to learn a new trade or develop a new skill. The work we are doing at forty may be too strenuous for us after we pass fifty. Or, we may develop a profitable avocation that will help us fi-

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nancially when full-time work is no longer practical. Many people have done this. It is during the middle years of life that we should begin planning a work reduction program—*not* a program of work elimination.

* * *

Another resource we must plan for is friends—just plain, old-fashioned friends. You know as well as I that you don't acquire *them* in a hurry. You have to cultivate them over a long period of time. The time to build up a wide circle of friends is while you are still young and active, so that as the years take their toll, some will still be left when you, yourself, are old. Friends are costly. They sometimes interfere with our pattern of living. They borrow—sugar, cigarettes, and even money. Sometimes, they don't return them. But they give us something, too—something we need more and more after our hair has reached the greying stage. They give us companionship and a helping hand when trouble strikes. We must plan well ahead of time, if, when we are old, we are to have friends and hold them.

* * *

Loneliness in old age is a more potent threat to women than it is to men. Most married women, by the time they reach the age of 65, will be widows. For better or worse, their husbands will be dead. You have probably heard the popular theory that attempts to explain why women outlive men and have amassed 80% of all of the money in the country. The theory is that men work so hard, and worry so much, trying to get enough money to take care of their wives after their own death that they drop dead from overwork. Be that as it may, loneliness during old age is a very concrete problem that many women are called upon to face.

A long and lonely life is not something any of us looks forward to with any degree of anticipation. That is why it is far more important to concentrate on the *quality* of life rather than on its *quantity*.

That is particularly true if we realize that we can *improve* the quality of life, but can do little to increase its quantity, as far as the oldsters go. You see, only children and young adults have had their lives extended appreciably during the past fifty years.

Only 1½ years have been added to the oldster's life span since 1900. That isn't enough to get excited about. It won't be until we know more about the fundamental causes of aging, and have developed means of checking the chronic diseases more effectively, that we will be able to add years to the life span of people over 65. So—we can't expect to live to be 100!

That's the story of aging, as I see it. Aging can be a great and stimulating experience if we want it to be—a bit like going down to the sea in a ship. When the winds blow and the seas begin to rise, the boat will sink or float, depending upon the sturdiness of the hull, the skill of the skipper, and the will of Providence.

We in the middle-aged group can see the storm clouds gathering up ahead. Whether *we* sink or float will depend upon whether we take steps to repair our aging hulls; orient ourselves to the blow ahead; and try more earnestly to enlist the support of Providence.

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GASTRIC SURGERY IN A SMALL HOSPITAL*

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The initial treatment of peptic ulcer is ordinarily a medical problem, consequently, the surgeon rarely sees a patient before he has developed one of the complications. It has been our desire to follow the patients who have undergone gastric surgery and who have, by the nature of their work (physical labor in most cases), been unable to follow a strict dietary management. Because of this fact, we often encounter patients who might have avoided surgery had they consulted their doctor earlier.

Some of the so-called complications of peptic ulcer are indications for surgery, and may be generally divided into four categories:

1. Perforation, which is one of the most spectacular and dramatic of abdominal catastrophies. Emergency laparotomy is ordinarily indicated so that surgical repair may be carried out. For many years it was thought that simple mechanical closure of the ulcer, with or without an omental graft as advocated by Graham, was the treatment of choice. However, in recent years two alternative modes of therapy have been advocated. These include the administration of massive doses of chemo-therapeutic agents and institution of constant gastric suction. This method has been advocated by British and French surgeons and has received support in this country by several clinics. We believe it should be reserved as a treatment for those cases that are such poor operative risks that laparotomy is inadvisable. In our own cases we have employed suction in only the advanced cases of peritonitis.

The other method is directed at ablation of the disease itself, as well as to control the acute emergency. Subtotal gastrectomy has been proven to be a safe procedure at time of surgery for the many acute perforations.

2. The second indication for surgery is hemorrhage from an active peptic ulcer. This may be indicated because of repeated small hemorrhages from an apparently refractive ulcer, or it may be in the patient who develops a massive hemorrhage. In these cases often the administration of blood cannot keep pace with the exsanguinating gastro-intestinal lesion. We feel that any patient over 45 years of age who has had repeated hemorrhages from peptic ulcer should undergo gastrectomy. It is usually wise to wait until the hemorrhage ceases and the blood volume has reached its equilibrium.

The decision as to when to operate upon a person with a massive gastro-intestinal hemorrhage is as difficult a problem as is found in surgery. Most of the patients are too sick for proper thorough examinations to locate the bleeding point. In certain cases it is practically impossible to distinguish between hemorrhage from esophageal varices, peptic ulcer, severe gastritis and a lesion located in the proximal small bowel. The use of the esophagoscope and a trial of therapy with the Blakemore tube have been of value in ruling out esophageal lesions.

Once the diagnosis of peptic ulcer has been made as the etiological factor of the hemorrhage, the optimum time for surgery is often a difficult problem. We have no direct rule that we follow, but often use as a guide repeated blood studies and hematocrit determination. If we are unable to keep the blood volume at a safe level with the transfusion of approximately 500 cc. of blood each 8 hours, we believe that laparotomy should be performed, with a great quantity of blood available, together with equipment to administer it rapidly.

3. The third indication for surgery is the patient with pyloric obstruction, or the so-called "blocked stomach". This is ordinarily the end result of many years of peptic ulcer disease and usually is accompanied by varying degrees of gastric dilatation and early emaciation.

4. The fourth surgical indication is usually designated as intractibility; ulcer symptoms which have persisted for years, and each exacerbation is worse than its predecessor. Often change of the type of pain will indicate penetration. Pain is usually refractive to even the most strenuous medical ulcer management, so that, conservative methods having failed, surgery is indicated.

*Read before the annual meeting of The Medical Society of Virginia at Virginia Beach, October 7-11, 1951.

In addition, the gastric ulcer we designate as the added indication. We also believe that all gastric ulcers are suspicious of malignancy. Carcinoma of the stomach, if it is to be cured surgically, must be treated early. The diagnostic facilities which we have at our disposal are not foolproof. Gastroscopy is still limited for effective use to certain large medical centers, and the majority of diagnoses are left to the Radiologists. The so-called observation period of medical management to observe the gastric ulcer crater for increase or decrease in size has been shown by Drs. Lahey and Cole to offer erroneous information as malignant ulcers will heal to some degree on medical management, with a decrease in size of crater and relief of symptoms, thus masking for several months the early growth of the cancer.

We do not propose to enter into a discussion as to the etiological relationship of a benign to a malignant ulcer, but we would like to emphasize that the gross appearance of the two may be so similar that the true diagnosis may not be discovered even after frozen section, but must wait the more accurate paraffin sections.

If this is true, it can readily be seen how helpless we are to accurately determine its malignant qualities by means other than surgical exploration in the suspicious cases. It has been observed by Allen that when resection is performed on the "cancer ulcer" group, the five-year survival was 40%, as compared with the 20% survival in the "gastric cancer" group. The Radiologist, adept as he is in differentiating benign from malignant lesions, calls a malignant ulcer benign in 10% of cases.

Since Billroth first suggested partial gastrectomy in 1881, surgical procedures upon the stomach and duodenum have taken many and varied forms. Billroth soon modified his technic from a simple resection and gastro-duodenostomy (Billroth I) to a resection with closure of the divided duodenum and anastomosis of the stomach pouch and proximal jejunum. Surgeons such as Polya, Hofmeister, Balfour, Horsley, Finsterer, Babcock and many others have perfected modifications of these two types of resection; and each has its own advocates who report satisfactory results.

Procedures of lesser magnitude than gastric resection have had their era of popularity, and certain of these limited procedures still may be indicated in selected cases. These include pyloroplasty, simple gastroenterostomy, excision ulcer, ligation blood supply, and, most recently, vagotomy.

The amount of stomach that should be resected has often been a point of arbitration. Several years ago it was generally believed that the more stomach resected the greater the chance of a permanent cure. However, now the pendulum appears to swing back toward a more conservative resection of 60-65% of the stomach. We believe if an excessive amount of gastric tissue is removed, the ability to resume a normal digestive process is endangered. Failure to gain weight following resection and the dumping syndrome are often attributed to removal of excessive amounts of the stomach.

The most popular type of anastomosis performed in this country at the present time is some modification of the Billroth II technic. In recent years the antecolic anastomosis is often preferred because of the greater facility with which it can be accomplished, and it is said to have no untoward effects as was originally thought. We would like to point out the fact that the Billroth I anastomosis should not be forgotten, as it can, in many cases, be more simply performed and restores the gastro-intestinal tract to a more normal physiological position. The chief criticism of this procedure has been that an adequate amount of stomach cannot be removed. We have found that in many individuals (particularly gastric ulcers) that 60-70% of the stomach can be resected; and the duodenum can be mobilized so that a tension-free anastomosis can be effected. We routinely do what we believe is an adequate resection and then decide upon the type of repair that may be performed. In 27% of our cases a Billroth I anastomosis was performed. Although the majority of Billroth II type of repair that we have performed have been retro-colic, we have no objection to an ante-colic method and are now employing it more and more frequently.

Pyloroplasty has been occasionally performed in the past for stenosing lesions from scar tissue in the pyloric regions.

In certain poor risk patients with pyloric obstruction, gastroenterostomy may be the procedure of choice as it is a much less traumatic operation.

Vagotomy has been used by us only in recent years, and except for one case, is not included in this series. We reserve its use for marginal ulcers.

Several years ago our attention was focused on

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the problem of recurrent perforations of the peptic ulcer. One case that came to us had undergone closure for a perforated ulcer twice in the past 18 months; perforated the third time, and, on exploration we found such a large callous ulcer that we believed a simple closure a mechanical impossibility. A gastric resection was performed, and the patient got along so well that we decided to investigate the possibilities of this procedure further.

European surgeons have for years performed gastric resection for treatment of perforation. Judine, Odelburg, Mulleder, Munberg and others have reported mortalities from 4 to 14%. An Italian surgeon, A. Franchini, reports 98 resections in a series of 200 cases of perforated ulcers, with a mortality of 10.3 for the resections, and 17.6 for the closures. Continuing our interest in this technic, Dr. Emmett reported our first seven cases of gastric resection for perforated ulcers at the Southern Surgical Association in December, 1948. Two of these seven cases were proven histologically to have been adenocarcinomas, unrecognized grossly at surgery. Up to the present time we have now resected 24 perforated ulcers without a mortality. Six of these have been for walled-off perforations, and the remaining perforations have been present for from 3 to 18 hours. We have found that the morbidity is less than for a routine resection, and there has been only one patient who has not been relieved of ulcer symptoms. We now employ resection as the treatment of choice in perforated ulcers, especially gastric ulcers.

Post-operative care of the patients undergoing gastric surgery includes carefully regulated fluid balance and blood transfusions. In an attempt to reestablish the normal physiological activities of the gastrointestinal tract, we remove the gastric tube as early as the day of surgery. The patient is started on oral liquids 48 hours post-operatively. However, close attention is paid to early signs of gastric dilatation and the tube is reinserted if indicated. Routine prophylactic chemo-therapeutic agents of sulfadiazine, streptomycin and penicillin are employed in various combinations.

In our series of 173 operative procedures upon the peptic region for ulcers, we found that 154 patients were males and 19 females. Initial symptoms of peptic ulcer disease were present for over five years in 90, or 52%; while 22% had had symptoms for 1-5 years. The precipitating or immediate symptoms

173 CASES OF GASTRIC SURGERY
1946-1950

Males	154
Females	19

MEDICAL MANAGEMENT

None	58
Less than six months	32
Six to twelve months	16
One to five years	25
Over five years	40

which demanded surgical relief were present for less than six months in 140, or 81% of the cases. 33% of the patients had not been on medical management prior to surgery. 18% had received medical management for less than six months; and 38% had been treated for over one year. Of the 173 patients seeking relief, 40, or 23%, had had previous gastric surgery. Closure of a perforation was most frequent, having been performed in 19 patients; seven had

PREVIOUS SURGICAL PROCEDURES	40
Closure of perforation	19
Gastroenterostomy	10
Gastric resection	7
Pyloroplasty	2
Excision of gastric diverticulum	1
Excision of peptic ulcer	1

had previous resections; ten had undergone gastroenterostomies; two pyloroplasties; one excision of a gastric diverticulum; and one excision of a peptic ulcer.

Indication for surgical intervention was called for by hemorrhage in 44 instances, or 28%; obstruction in 40, or 23%; perforation in 39, or 22.5%. Marginal ulcer, with intractable pain, was present six

INDICATIONS OF SURGERY

	No. Cases	
Hemorrhage	44	28 %
Obstruction	40	23 %
Perforation	39	22.5%
Suspicious gastric ulcer	30	17.2%
(Proven malignant)	10	
Intractable	13	7.0%
Marginal	6	4.0%
Congenital atresia	1	.6%

times; and in 30 instances a suspicious gastric ulcer, which could not be identified as cancer pre-operatively, constituted the indication. Ten of these, or 33%, were found to be malignant at time of surgery. Thirteen patients who were not responding

to prolonged medical management were operated upon. One patient had a congenital duodenal stenosis.

The types of procedures employed include 131 subtotal gastrectomies, or 75% of the cases. A Billroth I repair was done 33 times, or 27%. Eight had an ante-colic Hofmeister modification and 88, or 51%, had a post-colic Hofmeister anastomosis; 17 perforated ulcers were closed, and two perforated

TYPE OF SURGICAL PROCEDURE

Gastric resection	131
Post colic Hofmeister	88
Billroth I	33
Ante colic Hofmeister	8
Resection plus closure of colon fistula	2
Closure of perforated ulcer	17
Gastroenterostomy	16
Pyloroplasty	5
Drainage of abscess of perforated ulcer	2
Closure of gastroenterostomy	1
Vagotomy (abdominal)	1

ulcers with abscess formation were merely drained. 16 posterior gastroenterostomies were performed, and one patient had an old gastro-enterostomy taken down. Two patients underwent resection and closure of gastro-colic fistulae, and one abdominal vagotomy was performed.

PATHOLOGY

Duodenal ulcer	88
Gastric ulcer (benign)	48
Malignancies	15
Marginal ulcer	11
No ulcer (gastritis)	3
Inflamed intestine	1
Scar tissue	5
Hypertrophied pylorus	1

51% were duodenal ulcers. 48, or 27%, were benign gastric, and 15 malignancies, which were not diagnosed prior to surgery, but underwent surgery for either hemorrhage, perforation, or as a suspicious gastric ulcer, are included in the series. Eleven marginal ulcers were present, and in three

POST-OPERATIVE MANAGEMENT

	No. Cases	
Chemo-therapy		
Penicillin	110	
Sulfa	159	
Streptomycin	82	
Gastric suction	115	63%
Abdominal drainage	127	73%

instances, no ulcer was found in the resected specimen. Inflamed tissue was described once, and also one hypertrophied pylorus.

Post-operatively, penicillin was administered to 110 patients; sulfa to 159 (in wound and/or parenterally); and streptomycin to 82 in various combinations. Wangensteen suction was employed after the operative day in 115, or 63% of cases, and 73% of the cases had abdominal drains.

UNFAVORABLE RESULTS (RECURRENCES)

	No. Cases	%
Gastrectomy		
Diagnosis of recurrence	11	8.5
Required surgery	7	5.3
Closure Ulcer		
Diagnosis of recurrence	6	35.0
Required resection	4	23.5
Gastroenterostomy		
Diagnosis of recurrence	3	18.7
Required resection	2	12.5
Pyloroplasty		
Diagnosis of recurrence	2	40
Required resection	2	40

Follow-up studies have revealed 11 cases of resection who were diagnosed as having a recurrence. Seven, or 5.3%, required further surgery. Of the perforated ulcers closed, 10 have had recurrences; and four, or 23.5%, have already undergone surgery. Three of the 16 gastro-enterostomies have developed marginal ulcers, two necessitating resection. Two of the five pyloroplasties have undergone further surgery.

COMPLICATIONS

	No. Cases	%
No complication		79.2
Pneumonia	12	6.9
Wound infection	8	4.6
Wound separation	5	2.8
Delayed stomal opening	3	1.7
G-I fistula	2	1.0
Thrombophlebitis	2	1.0
Pancreatic fistula	2	1.0
Pelvic abscess	2	1.0
Dumping Syndrome	1	0.6

79.2% of the patients had no complications, but 12 patients, or 6.9%, had clinical evidence of pneumonia. Eight, or 4.6%, had wound infections; and five had wound separations requiring further closure. Three patients had edema of the stoma with gastric

retention over a longer than normal period. G-I fistula, thrombophlebitis, pancreatic fistula and pelvic abscess were found in two cases each. There was one patient with symptoms of a dumping syndrome.

Five deaths occurred in the 173 cases for an overall mortality of 2.8%. Four of these followed gastrectomy, or 3.0%. A summary of these cases is as follows:

Case A—A pulmonary embolus caused the death of an 18-day post-operative patient convalescing from a pyloroplasty.

Case B—Was operated upon for a massive G-I hemorrhage and underwent resection of his stomach. However, the bleeding continued, and patient expired on the second post-operative day despite massive replacement therapy.

Case C—Underwent a gastric resection for a suspicious gastric ulcer. The ulcer was malignant, and he died on the ninth post-operative day of peritonitis from a leaking suture line.

Case D—Age 74. Had a Billroth I resection for a bleeding duodenal ulcer and expired suddenly on the ninth post-operative day from a suspected pulmonary embolus.

Case E—Had undergone two previous procedures on her stomach for ulcer disease because of a massive hemorrhage from a marginal ulcer. The technical hazards were many, and post-operatively she developed an abdominal abscess which later required drainage. She left the hospital for one month and later returned with multiple abdominal abscesses and small bowel fistulae.

The average number of post-operative hospital days was 14.39 for all cases. For elective gastrectomies—14.8 days. For simple closure of perforated ulcer the average post-operative stay was 13.5 days; and in our selected group of 24 cases of gastrectomies done at time of perforation, the average number of post-operative days was 10.8.

DEATHS—POST-OPERATIVE

MORTALITY: ALL PROCEDURES	2.8%
GASTRECTOMIES	3.0%

1. Pyloroplasty, P.O. 18; pulmonary embolus.
2. Gastric resection, P. O. 2; continued massive hemorrhage. No ulcer found in specimen.
3. Gastric resection, P. O. 9; peritonitis, secondary to ruptured suture line.
4. Gastric resection, Billroth I; pulmonary embolus P. O. 9.
5. Gastric resection, 3 mos. P. O.
   Abdominal abscess with multiple small bowel fistulae.

AVERAGE NUMBER POST-OPERATIVE HOSPITAL DAYS

All procedures	14.39%
Gastrectomies	14.8
Closure perforation	13.5
Gastrectomy at time of perforation	10.8

In conclusion, we should like to repeat that we believe (1) subtotal gastrectomy is the treatment of choice for peptic ulcer disease complicated by hemorrhage, obstruction, intractability, and suspicious gastric ulcers. (2) 33% of the 30 suspicious gastric ulcers in this series were found to be malignant. (3) The Billroth I repair must not be forgotten. (4) We believe that in selected cases gastric resection is the preferred treatment for a perforated ulcer. In our cases the morbidity was less and the mortality nil.

A REVIEW OF PRESENT CONCEPTS ON THE AUTONOMIC CONTROL TO THE GASTROINTESTINAL VISCERA: CLINICAL APPLICATIONS*

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An increasing amount of knowledge has been gained in recent years concerning the autonomic innervation to the digestive tract, and in some measure has been gained through study of the effects of relatively new surgical procedures involving denervation of various segments of the sympathetic and parasympathetic pathways. In addition, the chemist and physiologist have broadened the scope of our concepts by indicating the chemical structure and mode of action of various neurohumeral substances through which these nervous effects, either excitatory or inhibitory, are mediated. This knowledge has been also enhanced by the use of certain inhibitors or blocking agents with anti-cholinergic or anti-adrenergic action which have become useful not only therapeutically but also as pharmacologic "tools" in the study of physiologic mechanisms.

Since there is a close interrelationship of cortico-hypothalamic centers, the pituitary gland and the autonomic nervous system, a resume of a few pertinent anatomic features will point out the role assumed by higher centers in the control of vegetative function.

The visceral nerve supply consists essentially of two contraposed systems, the parasympathetic and sympathetic, which, however, are not necessarily antagonistic. The integration of their action is through the central nervous system either by internuncial neurons in the cord or higher up in the medulla or hypothalamus, the former composing the simplest visceral reflex arc and the latter two more complex. The lateral and posterior hypothalamic nuclei are said to integrate sympathetic activity[1], while the midline and anterior nuclei control parasympathetic function[2]. Other nuclei regulate the posterior pituitary. Direct nerve connections with the anterior pituitary may exist although it has not yet been demonstrated, but it is known that stimulation of hypothalamic nuclei controlling the sympathetics will produce massive adrenergic discharge, which in turn causes increased activity of the anterior pituitary, with subsequent effects that are infinite.

At the highest integrating level, the cortex mediates partly the control of cardiovascular, gastrointestinal and glandular activities and at the same time is the area of conditioned reflexes, tensions such as worry, frustration and guilt, and crises such as rage, fear and certain compulsive phobias[3-6]. It is therefore possible to see that many visceral responses, either transient or sustained, may well be initiated in this sphere.

Consideration of the visceral nerve supply involves two groups, the motor or effector component and the sensory afferent pathways. The anatomical arrangement will not be further discussed except to mention that the motor outflow tract of both the sympathetic and parasympathetic systems consists of a pre- and postganglionic neuron. Both cranial and sacral parasympathetic postganglionic neurons lie in or near the wall of the organ innervated, while in the sympathetics, they are situated in the collateral ganglia (celiac, hypogastric, mesenteric) for the most part, and to a lesser extent in the paravertebral chain ganglia. Sympathetic visceral afferent fibers have cell bodies in spinal dorsal root ganglia, but their axons course through the various nerve trunks (e. g., splanchnic) and ganglia (e.g., celiac) alongside those of the motor fibers. Vagal afferent fibers have their cell bodies in the nodose ganglia which send fibers centrally to the sensory nucleus of the vagus.

In this connection, there have been repeated hypothetical explanations in the literature for the relief of peptic ulcer pain following vagotomy or after therapy with various anti-cholinergic blocking drugs. Among the more frequent is reference to the possible severance of pain pathways or to pharmacologic block of afferent pain fibers. Neither explanation is tenable if one means true visceral pain, for it has been conclusively shown that such impulses are mediated through either the splanchnic or hypogastric

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(sympathetic) pathways[7]. Since afferent pain fibers only course through the collateral and chain ganglia, they would normally not be subject to the blocking action of the usual anti-cholinergic agents. More logical reasoning would suggest atony (with the consequent buffering effect of food) plus secretory depression as the responsible factors[8].

This view is also in accord with the relief that is afforded in simple dysmenorrhea by anti-cholinergic drugs and by resection of the hypogastric plexus and is the natural sequence of the division of afferent pain tracts.

Although anti-cholinergic drugs may be beneficial in chronic pancreatitis, it is not by interference of pain pathways but by any of the following mechanisms: reduction in tone of the sphincter of Oddi, reduction in gastric HC1 and its stimulating effect, or inhibition of the cephalic or vagal phase of pancreatic secretion. The intractable pain of chronic pancreatic disease can only be abolished by interruption of the splanchnics or the sympathetic chain from T9 through T12, if surgery is directed toward the autonomic nervous system. Pancreatectomy may also effect relief, but the mechanism is clearly not the same.

It has been erroneously stated that the performance of subtotal gastrectomy (three-fourths' resection) approximates at the same time a vagotomy by division of subserous vagal branches. It can be plainly seen that such is not the case when it is recalled that most of the posterior vagus shortly leaves the cardiac portion of the stomach just below the diaphragm and enters the celiac ganglion or plexus.

It appears that the parasympathetic and sympathetic systems are contraposed but not always antagonistic since the vagus is cardio-inhibitory and visceromotor and the sympathetic cardio-excitatory and viscero-inhibitory[7]. For blood vessels to the viscera, the vagus is vaso-inhibitory and the splanchnics vasoconstrictor[1]. The above statements apply only to motor function. For secretory function the vagus is excitatory while the sympathetics are trophic, but not inhibitory; that is they induce the formation of precursor secretory granules within the gland cell.

The pathogenesis of peptic ulceration has long been thought to be intimately related to autonomic nervous function and several factors have been variously ascribed to be of pre-eminent importance: gastric acidity, peptic activity, muscular spasm and ischemia of vascular origin. It seems likely that multiple derangements in function of the stomach and duodenum are simultaneously operative and that ulcer formation is possible only when two or more of these functional aberrations are present, for instance, ischemia + acid + pepsin. The threshold for ulcer formation may thus depend upon the degree of overactivity or imbalance, either relative or absolute, of these basic functions which are largely controlled by autonomic nervous outflow. The elements of hypersecretion and motility have been extensively studied in the past. In addition, recent significant work upon the vascular element by Barclay and Bentley[19] revealed the existence of submucosal vascular shunts in the stomach which become manifest as a response to (operative) trauma not only to the stomach but, also more significantly, to other body sites, and can be prevented by sympathetic block or spinal anesthesia. The mucosal ischemia shown by Barclay would be similar to that noted in the renal cortex with the so-called Trueta shunt. About the same time, other investigators reported prevention of duodenal ulcers by sympathetic block prior to the injection of a stressor agent (dysentery toxin) intravenously into dogs[20], while ulceration almost invariably occurred without a preceding block.

For the sphincters the motor situation is rather the reverse of that present elsewhere in the G.I. tract and for very sound physiologic reasons. The cardia, pylorus, rectal and internal anal sphincters are maintained in tonic contraction by sympathetic impulses, which also promote a state of decreased tone in the body of the viscus concerned. By comparison, the opposite effect occurs with vagal stimulation just as in the stomach, peristalsis is initiated and the pylorus relaxes to empty the stomach, the stimulus to relaxation being an oncoming contraction wave. The ileocecal sphincter deviates from this rule and has been found to be relaxed by adrenine or sympathin-like substances.

Perhaps the least understood segment of the visceral autonomic system is the parasympathetic network of postganglionic neurons and nerve plexuses (submucosal and myenteric plexuses of Meissner and Auerbach). It has been mentioned that for the cranial outflow tract (vagal) these postganglionic neurons are quite short and located in the wall of the particular viscus. These plexuses seem quite capable of acting as intrinsic nerve nets with autot-

omous activity when removed from the control of a preganglionic motor fiber such as the vagus nerve. Assumption of such function may explain the gradual restoration of motility and basal secretory activity after vagotomy even though the cephalic stimulus to secretion and motility has been abolished, as evidenced by a lack of response to hypoglycemic stimulation of the vagus. Even with severed preganglionic vagal trunks, these "abandoned" or isolated postganglionic neurons seem to acquire later an intrinsic spontaneous activity after some months[7].

Recently there has appeared more evidence to substantiate a congenital or acquired absence of the myenteric ganglia of Auerbach at the cardia and lower colon in those cases of cardiospasm, megacolon and Hirschsprung's disease[12,18]. In such areas there may be a failure in relaxation, thereby creating a block to peristaltic progression by a segment of tonically contracted smooth muscle. This has been shown to be a characteristic sequence of denervation of smooth muscle in lower vertebrates[18]. Resection of the involved segment has been reported curative in megacolon[13], in contrast to the previous unsatisfactory results with sympathectomy. A comparable "splitting" of muscle fibers may perhaps occur with bouginage or hydrostatic dilatation in the treatment of cardiospasm. Recently topical procaine (by ingestion) has been shown to be effective for several hours in the relaxation of the tonic segment of cardiospasm possibly by interrupting transmission at effector nerve endings or by direct action upon smooth muscle[21].

As an integral part of the sympathetic system it is interesting to note the special anatomic and functional adaptation of the adrenal medula. Here there is no lengthy postganglionic sympathetic nerve, but the preganglionic sympathetic nerve endings terminate directly upon the medullary cells. Significantly, the medullary cells secrete adrenergic substances similar to those liberated at other visceral postganglionic sympathetic endings. This intriguing fact is not coincidental but probably due to the embryologic origin of the medullary or chromaffin cells which arise from sympathetic neuroblasts just as do the sympathetic chain ganglion cells, which are postganglionic neurons. Adrenal medullary cells may therefore be thought of embryologically as counterparts, in this location, to the postganglionic sympathetic neurons in chain and collateral ganglia. With the knowledge that both medullary cells and postganglionic sympathetic endings liberate adrenergic substances, the developmental point just mentioned indicates that chromaffin cells of the adrenal medulla are uniquely adapted postganglionic neurons in essence, and are supplied with a rich vascular network for a specific function, that is the rapid liberation of adrenergic substances into the systemic circulation for widespread and immediate effect (DeGaris' concept)[7].

A discussion of cranial-sacral outflow tract would not be complete if the controversy over the extent of the vagus were not mentioned. The older workers believed the vagus innervated the colon to the level of the distal transverse colon. However, it has been found that vagal fibers supply the ascending colon, to a lesser degree the transverse, but not the descending; while the sacral parasympathetic nerves supply the descending and sigmoid colon, to a lesser extent the transverse, but not the ascending[11]. This arrangement provides a broad zone of overlapping between the cranial and sacral parasympathetics and provides the colon with an arrangement suited better for smoothly integrated function by a gradual transition of control over the intramural myenteric nerve plexuses and nerve nets (postganglionic neurons).

This brings us finally to the neurohumeral effector substances liberated at both pre- and postganglionic nerve endings. The chemical mediator is acetylcholine at all preganglionic endings of both systems and also at parasympathetic postganglionic endings. It is also found at somatic myoneural junctions and at synapses within the substance of the central nervous system.

Recently it has been discovered that cholinesterase (including cholinesterase-like substances) varies at these particular sites both in amount and types[15]. That is significant, for it may explain why certain pharmacologic blocking agents have selective affinity for organ systems (Banthine on alimentary tract[16]) for autonomic ganglia alone (C_6—or hexamethonium[17]) and others have a curariform effect upon striated muscle (C_{10} compounds)[17]. The development of newer and more efficacious blocking agents in the future may depend upon the final elucidation of the chemical nature, specificity and function of these cholinesterases.

An adrenergic agent, sympathin, closely related to epinephrine and neo-epinephrine, is the effector

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substance at postganglionic sympathetic nerve endings with the exception of sweat glands.

When estimating final results, it can be seen that the action upon any one visceral organ is determined first by the type of organ (gland or hollow viscus) and the degree of integrated activity in force as compared to that existing normally between the two systems and the net result of these forces. Acetylcholine and the parasympathetics are visceromotor, vasodilator and secretory and adrenergic substances and the sympathetics are viscero-inhibitory, vasoconstrictor and non-secretory (but trophic). In intact man no one component can operate to the total exclusion of contraposing forces.

The pharmacologic effects of neurohumeral substances may be better understood by considering the anatomic location where liberated and by noting the effect of various so-called blocking agents or antagonists. This subject is too extensive except to mention that it should be kept in mind that preganglionic endings in both systems are cholinergic and will both be blocked to varying degrees by anticholinergic drugs. Also agents known as choline esters (urecholine and acetyl-B-methylcholine or mecholyl) will substitute for the effect of acetylcholine at the preganglionic ending and will produce both parasympathetic and sympathetic visceromotor effects to varying degrees. Because cholinesterase destroys acetylcholine rapidly, the group of drugs which act by inactivating cholinesterase will also have some sympathomimetic besides parasympathomimetic effects (neostigmine). Thus in vagotomized patients neostigmine has a paradoxical effect and increases the gastric atony by allowing impulses to flow through the sympathetic chain ganglia unchecked. Atropine is said to act as a sympathomimetic drug by preventing the entrance of acetylcholine into the secretory cell[22].

Regarding adrenal medullary function as a whole, it may be said that adrenergic discharge produces a mass effect since epinephrine from the adrenal medulla reaches the systemic circulation in large amounts by way of its rich vascular supply and is only slowly destroyed by the oxidase or tyrosinase in the general circulation. In contradistinction, cholinergic discharge generally is confined and local effects predominate. This is because conditions are not anatomically and physiologically favorable for cholinergic substances to reach the general circulation since it is released only at the nerve endings, and to further insure only a local action, it is quickly destroyed before diffusion outside these confines would be possible.

Thus one system is adapted in DeGaris' terms, for "fight or flight, the other for conservation, digestion and restoration"[11]. This arrangement is admirably devised for preservation of a balanced organism during its response to changes in the external environment, thereby contributing to the many endocrine and nervous arrangements that enhance homeostasis.

SUMMARY

A review of currently held concepts on the anatomy and function of the autonomic nervous supply to the gastrointestinal tract is presented. Certain aspects that are pertinent to clinical problems are discussed.

REFERENCES

1. Ranson, S. W.: Trans. College of Physicians of Phila. **2**:222, 1934.
2. Wang, S. C.; Clark, G.; Dey, F. L., and Ranson, S. W.: Am. J. Physiol. **30**:81, 1940.
3. Alvarez, W. C.: An Introduction to Gastroenterology. 4th Ed. p. 268, Paul Hoeber, N. Y. 1948.
4. Kuntz, A.: The Autonomic Nervous System. 3rd Ed., Lea & Febiger, Phila. 1945.
5. Watts, J. W., and Fulton, J. F.: New England J. Med. **210**:883, 1934.
6. Thomas, J. E.: Gastroenterology **12**:545, 1949.
7. DeGaris, C. F.: J. Assoc. Am. Med. Coll. **25**:417, 1950.
8. Plummer, K.; Burke, J. O., and Williams, J. P.: Sou. Med. J. **44**:801, 1951.
9. Annis, D., and Hallenbeck, G. A.: Gastroenterology **17**:560, 1951.
10. Moore, R. M., and Singleton, A. O.: Am. J. Physiol. **104**:267, 1933.
11. Dragstedt, L. R.: Ann. Surg. **122**:973, 1945.
12. Whitehouse, F. R., and Kernohan, J. W.: Arch. Int. Med. **82**:75, 1948.
13. Swenson, O.; Neuhauser, E. B. D., and Pickett, L. K.: Pediatrics **4**:201, 1949.
14. Schmidt, C. A.: Proc. Soc. Exper. Biol. & Med. **30**:739, 1933.
15. Whittaker, V. P.: Physiol. Rev. **31**:312, 1951.
16. Longino, F. H.; Grimson, K. S.; Chittum, J. R., and Metcalf, B. H.: Gastroenterology **14**:301, 1950.
17. Paton, W. D. M.: Br. Med. J. April 4, 1951, p. 773.
18. Alvarez, W. C.: Gastroenterology **13**:422, 1949.
19. Barclay, A. E., and Bentley, F. H.: Gastroenterology **12**:177, 1949.
20. Penner, A.: Abstr. Program Am. Gastroenterological Assn. April 29, 1950 (pre-printed).
21. Balfour, D. C., Jr., and Wharton, G. K.: Gastroenterology **18**:606, 1951.
22. Goodman, Louis, and Gilman, Alfred: Pharmacological Basis of Therapeutics. The Macmillan Co., New York, 1941.

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BASAL CELL EPITHELIOMA IN CHILDHOOD*

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Basal cell epithelioma of the skin is essentially a neoplasm of middle age and later life, and although it has been stated that such lesions may occur at any age, they are distinctly uncommon among children and in the early decades of life. If examples of basal cell epithelioma associated with xeroderma pigmentosum are discarded, the incidence of this tumor in young people is low indeed. The case described below, recently encountered on the Dermatology Service of the University of Virginia Hospital, emphasizes the necessity of being aware that such lesions do occur in children.

REPORT OF A CASE OF BASAL CELL EPITHELIOMA IN A GIRL AGED 14

M.H., a 14 year old white female, was first seen in the University of Virginia Hospital on September 4, 1951, complaining of a small "sore" on the face below the right eye. She stated that it had been present for eighteen months and had been slowly enlarging in spite of various types of local therapy, including antibiotic ointments.

Examination revealed a small ulcerated lesion approximately 1 cm. in diameter over the right malar region just below the outer canthus of the right eye (Fig. 1). It was

Fig. 1—(patient M.H.).—Lesion of right malar region, of 18 months duration.

roughly oval in shape with an elevated, rolled pearly border which exhibited a number of telangiectatic blood vessels. There was a central crater which was somewhat depressed and moist. There was no evidence of erythema or obvious infection. The remainder of the patient's skin was normal. The clinical impression was basal cell epithelioma. The patient was referred for surgical excision and microscopic study of the excised tissue.

She was admitted to the University of Virginia Hospital as an in-patient on September 18, 1951, and excision carried out. A full thickness skin graft was used to cover the area. She was discharged in eight days, the graft having taken perfectly. The pathologic report stated that the tumor had been totally removed.

Microscopic examination of the section revealed destruction of the epidermis in the central area of ulceration. Immediately below this ulcer were seen variously sized and shaped masses of "basal cells" with the peripheral cell layers of the masses showing palisade arrangement of nuclei (Fig. 2). Islands of the tumor were seen invad-

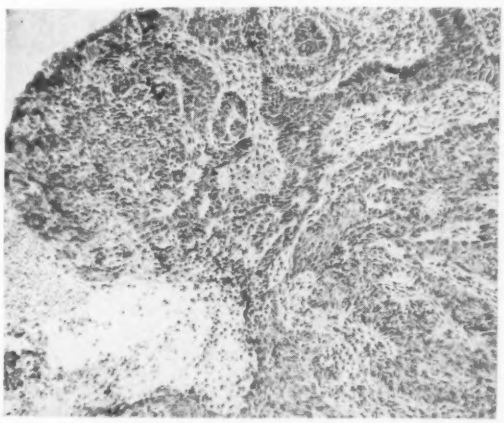

Fig. 2—(patient M.H.).—Low-power view of section from excised lesion.

ing deeply into the corium. In the deeper portions of the lesion there was a slight tendency toward keratinization and abortive attempts at pearl formation The histologic diagnosis was basal-cell carcinoma with a slight admixture of squamous-cell elements.

DISCUSSION

There is general agreement that basal cell epithelioma is a disease of advanced years and is rarely observed in children. Lesions of this type, however, are occasionally seen during early life and this fact is mentioned in several texts. Kaposi[1] acknowledged the predisposition of advanced age but stated that

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he had seen epithelioma in several patients between the ages of eight and 18 years. He failed to qualify the cases further in regard to type or the presence of a pre-existing dermatosis such as xeroderma pigmentosum. Mackee and Cipollaro[2] also refer to personal observations of a "number of cases in patients between 16 and 22" and illustrate a case of basal-cell epithelioma of the lip of a girl aged 22 with the comment that this was unusually young for epithelioma to develop.

There have been few case reports in the literature citing specific examples of basal-cell epithelioma in childhood or adolescence and various authors have made statistical analyses of large series of cases of cutaneous cancer which indicate that primary basal-cell epithelioma is indeed a rare occurrence prior to adult life.[3,4,5,6,7,8] Sequeira[9] in 1912 recorded a case of rodent ulcer on the back of a 12 year old boy which was unusual from the standpoints of both age and location. This patient, however, gave a history of having had a "mole" at the site of the lesion since the age of two. Owen,[4] in the course of reviewing the microscopic sections from 836 cases diagnosed basal-cell carcinoma, noted that the youngest patient was 18 years of age. In another review by de Cholnoky[5] only one case below the age of 20 years was reported from a total of 1062 patients with cancer of the face. This lesion was present over the right parotid region of the face of a girl of 19. Zeisler[8] in 1933 failed to observe a single instance of basal cell epithelioma under 21 years of age in a series of 359 cases.

We feel it worth while to call attention to the fact that basal-cell epithelioma does occur in young persons and to point out that the diagnosis cannot be discarded on the basis of the patient's age alone. The child whose case is reported above had a basal-cell epithelioma of the skin which was typical from the clinical standpoint, was not secondary to a pre-existing dematosis, and resembled similar tumors seen in older subjects in every respect.

SUMMARY

1. A case of basal-cell epithelioma occurring on the face of a 14 year old girl is reported.

2. Attention is called to the fact that, although relatively rare, basal-cell epithelioma is occasionally encountered in the early years of life.

REFERENCES

1. Kaposi, Moritz: Pathology and Treatment of Diseases of the Skin for Practitioners and Students, Translated from the 5th German edition under the supervision of James C. Johnston, New York, William Wood & Co., 1895.
2. Mackee, George M., and Cipollaro, Anthony C.: Cutaneous Cancer and Precancer. Publication of The American Journal of Cancer, Lancaster Press, 1937.
3. Ward, Grant E., and Hendrick, James W.: Malignant Epithelial Tumors of the Skin of Head and Neck. Am. J. Surg. **79**:771-786, 1950.
4. Owen, May: Basal Cell Carcinoma. Arch. of Path. **10**:386-394, 1930.
5. de Cholnoky, Tibor: Cancer of the Face. Ann. of Surg. **122**:88-101, 1945.
6. Warthin, Aldred Scott: The Occurrence of Malignant Neoplasms in the Young. Arch. Int. Med. **15**:444-450, 1915.
7. Hagen, H. H.: Basal Celled Cancers of the Skin. So. Med. J. **10**:241-245, 1917.
8. Zeisler, Erwin P.: Cancer of the Skin. Surg., Gyn. & Obs. **56**:472-475, 1933.
9. Sequeira, J. H.: Rodent Ulcer of the Back in a Boy of Twelve. Brit. J. of Derm. **24**:391-392, 1912

RESULTS OF SUBTOTAL GASTRIC RESECTION FOR PEPTIC ULCER

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Subtotal gastric resection is today the most commonly used method of treating intractable or complicated peptic ulcer. In Norfolk General Hospital during the year 1940, only six gastric resections for peptic ulcer were done. In 1950 there were thirty-two resections, an increase of over 500 per cent. In all the civilian hospitals in Norfolk during the year 1950 there were sixty-six stomach resections done.

It is estimated that there are now living in the City of Norfolk between 300 and 500 patients who have had a subtotal gastric resection. How well are these people adjusted in their environment? How many are in good health and able to carry on their usual occupations?

In an effort to answer these questions, we have reviewed a group of our own patients for a five-year period—from 1946 to 1950. The reason for selecting this group is as follows:

1. They were all done by a similar method in civilian hospitals in Norfolk.
2. They have been followed for a sufficient period of time to gain a fair knowledge of their adjustments. Most of them have been followed for several years. A few have failed to report after the first year or so and may have developed complications not included here.

From 1946 to 1950, we have had sixty-one resections for peptic ulcer. One patient died eight days postoperatively, presumably from coronary thrombosis; but this must be classified as a surgical death. The surgical mortality was, therefore, 1.6 per cent. The sixty surviving cases are presented as the basis of this review.

The type of surgery which has been used is a modified Hofmeister resection in which approximately two-thirds of the stomach is removed (Fig. 1). The anastomosis is placed anterior to the colon, with a short proximal loop of jejunum. The usual indications were intractability, hemorrhage, perforation, or obstruction. Four cases were done for recurrent or jejunal ulcer after previous surgery. In three of these patients an insufficient amount of stomach had been removed at the original operations, which were done elsewhere. In one, a gastro-enterostomy had previously been done.

The important late complications observed were:

1. Dumping syndrome ----------7 patients
2. Post-resection gastritis --------4 patients
3. Hypoglycemia ----------2 patients
4. Hemorrhage ----------1 patient

(1) Dumping Syndrome

This is a phenomenon which has never been satisfactorily explained. Immediately or soon after

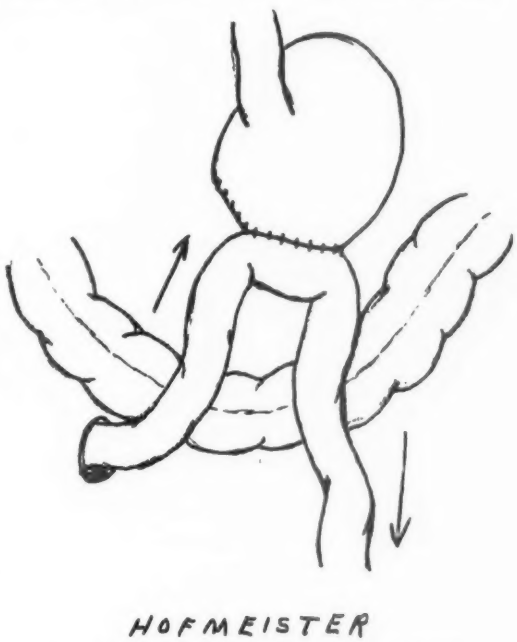

Fig. 1.—Modified Hofmeister resection placed anterior to the colon.

taking food the patient develops weakness, dizziness, sweating, palpitation, nausea, and is forced to lie down.

It occurs most often after breakfast; in fact, it is rarely seen after other meals. The symptoms are very similar to those seen in hypoglycemia and these two conditions have very frequently been confused. An early theory proposed that the rapid emptying of

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the stomach produced a sudden rise in blood sugar, which in turn stimulated an unusual mobilization of insulin. With the rapid rise in insulin, hypoglycemia developed, producing the symptoms described above.[1] More recent studies have shown that hypoglycemia does not develop immediately after eating, and that at the time of the attack blood sugar levels are normal or elevated.[2] Machella[3] has been able to reproduce the syndrome by experimentally inflating a balloon in the upper jejunum. He is of the opinion that the attacks are precipitated by hypertonic solutions of foodstuffs, drawing fluid into the lumen of the gut. This would explain why the attacks are more common after a high carbohydrate intake, especially when taken in liquid form.

Radiographically, practically all post-gastrectomy stomachs empty rapidly. Why only a few develop the dumping syndrome is unknown. Some other factor must play a part, possibly a vagotonia, since atropine alleviates the condition. The attacks can usually be prevented by eating only solid food at the beginning of a meal, by eating slowly, by using a diet low in carbohydrates, and by the use of atropine.

The attacks usually subside spontaneously in a few months and are rarely seen as late as one year after operation. While it lasts, it is a most distressing condition, and these patients should not be labeled psychoneurotic.

(2) Post-Resection Gastritis

Patients suffering from this condition complain of pain, nausea, nutritional and psychic disturbances. The symptoms are sometimes as bad or worse than the original ulcer, and the response to treatment is poor. The symptoms are made worse by over-eating and by alcohol. In spite of the achlorhydria which is usually present, therapy with hydrochloric acid usually makes the patient worse.

On an ambulatory ulcer type of diet, with vitamin reinforcement, most of them can return to work. They are, however, the most unhappy and dissatisfied group seen following this type of surgery.

Of the four patients who have had post-resection gastritis, only two have continued to have symptoms up to the present time. In these, the symptoms have existed for one and two years, respectively.

(3) Hypoglycemia

This condition, as stated above, is frequently confused with the dumping syndrome. The symptoms are similar, but the time interval is entirely different. Where the dumping syndrome appears directly after eating, hypoglycemia develops two to three hours later. The rapidly emptying post-gastrectomy stomach produces a rapid rise in blood sugar with unusual stimulation of insulin. Since the food passes rapidly through the intestinal tract, the buffering effect of continued absorption is lost. A rebound hypoglycemia subsequently develops with the usual classical symptoms. Why it should happen only in rare cases is unknown.

The treatment of hypoglycemia is much more satisfactory than in the dumping syndrome. A low carbohydrate diet with six small meals daily usually controls the symptoms. Once an attack develops, it is relieved almost immediately by ingestion of carbohydrate, such as candy, Coca-cola, or sweetened orange juice.

(4) Hemorrhage

One patient in this group developed a massive unexplained hemorrhage three months after operation. He required hospitalization and blood transfusions. X-rays were negative for jejunal ulcer, and there was no pain as would have been expected with jejunal ulcer. This hemorrhage was attributed to superficial erosion or gastritis, but the cause was not proven.

The patient subsequently returned to work and has had no recurrence of symptoms in the past four and one-half years.

Marginal or recurrent ulcer has been omitted from the list of major complications because there has been no proven case in this group, in so far as we can ascertain. In 1500 cases of subtotal gastric resection reviewed by Rindone,[4] 14 cases of recurrent ulcer were found, or .8 per cent.

The incidence of recurrent ulcer has been shown to have a definite relationship to the amount of stomach removed at operation. In the rare case that develops after an adequate high resection, vagotomy, is considered the treatment of choice.

In addition to the important complications outlined above, there are a group of lesser disturbances which should be mentioned:

1. Failure to gain weight.
2. Anemia.
3. Dyspepsia and regurgitation.
4. Food intolerance.

(1) Failure to Gain Weight. This is a very com-

mon complaint following gastric resection and occurs in over fifty per cent of cases. The etiology is probably related to the rapid passage of food through the intestinal tract. It occurs in spite of a large food intake. It apparently does not affect general health or ability to work.

(2) Anemia. This is almost invariably of the secondary hypochromic type and responds well to treatment. We have seen no case of primary anemia. Ivy[5] reported that in dogs subjected to total gastrectomy and observed for as long as ten years, no case of primary anemia developed.

(3) Dyspepsia and Regurgitation. This condition is occasionally seen and is probably related to the reduced size of the stomach, or the reflux of bile into the stomach. The symptoms are made worse by overeating, rapid eating, or by an intake of rich or greasy food. It is usually a time-limited condition which disappears in a few months after operation as adjustment takes place.

(4) Food Intolerance. In a few patients, following resection there is a selective intolerance for certain foods. Concentrated carbohydrates are not tolerated well and an aversion to candy is common. This, like many other post-gastrectomy complaints, tends to improve with time, and the patient who two months after surgery complains that he can not eat sweets, will two years later state that he can eat or drink anything.

One interesting complaint that is frequently noted is a profound aversion to milk. Even as late as five years after operation, an occasional patient will state that milk is extremely distasteful, even though he takes all other foods well.

SUMMARY

A critical review has been made of a group of patients undergoing subtotal gastric resection for peptic ulcer. While some of the results in this group are far from perfect, it must be remembered that surgery was performed only after all other methods of treatment had failed. This report has purposely brought out all of the known unsatisfactory features associated with gastric resection. Of the 61 patients operated upon, 54 or over 88 per cent are known to be working and have been relieved of all important symptoms.

BIBLIOGRAPHY

1. Glaessner, C. L.: Hygerglycemic Shock. Rev. Gastroenterol. **7**:528-533, 1940.
2. Culver, Perry J.: The Dumping Syndrome. Med. Clin. North America, Mass. Gen. Hosp. No. Sept. 1948:1321-1340.
3. Machella, Thomas E.: The Mechanism of the Post-Gastrectomy "Dumping Syndrome." Ann. Surg. **130**:145-159, 1949.
4. Rindone, Giovanni: Peptic Ulcer Following Gastric Resection. Surg. Gyn. and Ob. **87**:347-348, 1948.
5. Ivy, A. C., Richter, O., Meyer, A. F., and Greengard, H.: Relation of Gastrectomy to Anemia in Presence of Substances Effective in Pernicious Anemia in Canine Stomach and Liver. Am. Jour. Digest. Dis. and Nutrition, **1**:116-119 (Apr.), 1934.

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New Books.

Listed below are some of the newer books in the Tompkins-McCaw Library of the Medical College of Virginia. These are available to our readers under usual library rules.

Bland, John H.—The clinical use of fluid and electrolyte. 1st ed. 1952.

Bond, D. D.—Love and fear of flying. 1st. ed. 1952.

Brody, E. B., Redlich et al.—Psychotherapy . . . with special reference to schizophrenia. 1st. ed. 1952.

Ciba Foundation Conference—Isotopes in biochemistry. 1st ed. 1951.

Gesell, Arnold L.—The child from five to ten. 1st ed. 1946.

Greenblatt, R. B.—Office endocrinology. 4th ed. 1952.

Parkinson, Roy H.—Tonsil and allied problems, 1st ed. 1951.

Rehfuss, M. and Price, A. H.—A course in practical therapeutics. 2nd ed. 1951.

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CLINICOPATHOLOGICAL REPORTS

From the Case Records of the Medical College of Virginia and the University of Virginia Hospitals

HARRY WALKER, M.D., *Editor*
WILLIAM KAY, M.D., *Associate Editor*

CASE NO. A6768

A 36 year old, colored, female school teacher was transferred from a tuberculosis sanatorium to St. Philip Hospital on May 5, 1951, because of recent development of personality and neurologic changes.

She had had no previous significant illness and had been in good health until a routine chest x-ray in February, 1950, disclosed a "round density behind the 3rd rib on the left with linear streaking in the 1st and 2nd left interspaces". This was interpreted as probably tuberculous, but of questionable activity. It was learned that the patient's mother had died in 1937 with pulmonary tuberculosis, but this was the only known contact. Several sputum examinations at this time were negative and the patient was asymptomatic. She was followed as an out-patient for several months and repeated chest x-rays in April and July of 1950 were interpreted as showing no change. In January, 1951, however, progression of the lesion was noted and the patient was then admitted to the sanatorium, still asymptomatic.

At this time the general physical examination was negative except for a small left supraclavicular node. Hemoglobin, urine, and WBC were normal and the sedimentation rate was 20 mm/hr. Flocculation negative. OT 1:10,000 read as 3+ in forty-eight hours. Repeated sputum examinations were negative for acid-fast bacilli, including one culture. Chest x-ray reported as "enlarged hilar shadows on left; extending from this area into the 2nd and 3rd anterior interspaces is an exudative type of lesion".

Pneumothorax was instituted and the patient ran an uneventful afebrile course. She was cheereful, alert, and attended classes regularly in the sanatorium until around the first of April, 1951, when she began to show definite changes in her bahavior. Attendants felt that these changes began to develop after the patient witnessed the death of another patient from massive pulmonary hemorrhage. She became rather silly, untidy, and apparently unable to take her temperature or read the thermometer correctly. The patient herself began to complain of a "flickering in front of her eyes" and occasional stumbling and falling over objects.

On April 20, 1951, neuropsychiatric examination revealed the following: The patient answered questions vaguely and her mood was somewhat silly but not markedly inappropriate. She admitted some depression, mainly in regard to pulmonary disease. She was oriented as to person and place, but poorly oriented as to time and unable to give her age, date of birth, or to subtract serial sevens. The corneal and abdominal reflexes were absent and the deep tendon reflexes were hyperactive. She could not execute heel-to-toe walking or finger-to-nose test accurately, was unsteady on her feet, walking on a broad bases and tending to lean on attendant. No nystagmus or muscle weakness was noted and no sensory changes were demonstrable, though patient complained of tingling of extremities. Other than absent corneal reflexes, the cranial nerves were normal and the remainder of the examination was negative. A lumbar puncture was entirely negative.

She became progessively worse, developed a cough, and complained of severe headache after coughing, and was then transferred to St. Philip Hospital for further study.

On admission temperature, pulse and respiration were normal. Blood pressure 130/80. In addition to the findings noted above, the patient was found to have a rather fixed facies, a slow rhythmic speech, and a positive Babinski on the left. The remainder of the physical examination was negative except for evidence of left pneumothorax.

Laboratory Data: Urine negative. Hemoglobin 11.8 grams, WBC 14,600 with 88 polys. Repeated lumbar punctures showed no abnormalities except for some increase in pressure on third tap. Skull films showed slight thickening of the skull vault throughout with hyperostosis in the frontal, parietal, and occipital regions. A chest x-ray showed a left pneumothorax with some density of the left hilum.

Following admission the patient went steadily downhill, was incontinent, and vomited frequently the first few days after admission. She continued to

complain of headache after coughing. On May 28th a ventriculogram was attempted, but satisfactory filling of the ventricles was not accomplished. Before these studies could be repeated the patient became much worse, developed high fever, tachycardia and expired on June 3, 1951. An autopsy was performed.

DISCUSSION BY DR. E. S. RAY

Briefly, this case was a 36 yr. old colored female who had a routine chest x-ray in February 1950 that revealed a small infiltrative lesion in the left upper lung field. She had no symptoms at the time. Her mother had died of tuberculosis 13 years previously and apparently she had been x-rayed at intervals with the film of February 1950 being the first one to indicate any abnormality. It is understandable how, in view of the Family History of tuberculosis, such a lesion should be interpreted as tuberculosis. Sputum studies for tubercle bacilli were negative. She was followed as an out-patient with frequent x-rays for 5 months with no apparent change. In January 1951 (11 months after the first positive x-ray) there was a definite progression as shown by x-ray but she continued asymptomatic. Because of this x-ray change she was admitted to the sanatorium. Except for a small supraclavicular lymphnode, the physical examination was negative. This is not surprising since the chest film at that time suggested that the lesion was a peripheral mass of some nature and these produce few if any abnormal physical signs. A left lateral film would have been of value not only in localizing the lesion more accurately but also would have more definitely indicated whether this was a peripheral tumor mass or an infiltrative process in the lung — the P.A. film certainly suggests a tumor mass to me. The chest films also revealed that in addition to the pulmonary lesion there was definite enlargement of the left hilum which has the appearance of large lymphnodes. Routine laboratory studies were normal. She had a fairly strong tuberculin reaction but this is of little value in determining the nature of this mass except to indicate that it could be tuberculosis. Sputum studies were again negative for tubercle bacilli even by culture. Tuberculosis apparently was still the diagnosis of her physician and because there was progression of the disease, pneumothorax was induced and as can be seen a good collapse of the lung was obtained but the density in the lung did not collapse, nor did it change its size or shape. She continued to feel well until April 1951, three months after admission to the sanatorium, when she developed personality changes, complained of visual disturbances, and was noted to stumble and fall on occasions. She complained of headache on coughing and became progressively worse. Because of these changes, which indicated some organic brain lesion, probably a tumor mass, she was transferred to St. Philip. A lumbar puncture was negative except for an increase in spinal fluid pressure—additional evidence of an intracranial tumor mass of some nature. Her headache continued and she began to vomit. Her course was rapidly downhill and she died on June 3, 1951.

With the complete course of this patient's disease available, the diagnosis is obviously one of two diseases: She had either a peripheral bronchogenic carcinoma with metastases to the brain (frontal lobe and probably to the cerebellum), or she had a tuberculoma of the lung with a metastatic tuberculoma of the brain. To me carcinoma of the lung with metastases is the more likely.

I base this on the following facts:

1. The chest film in June 1950 and in January 1951 revealed a fairly well circumscribed lesion of the lung which failed to change either its shape or size following pneumothorax.
2. There were rather large hilar lymphnodes which are more frequently seen in carcinoma than in inflammatory lesions.
3. Evidence of C.N.S. metastases which is a frequent occurrence in primary pulmonary neoplasms and not so frequent in pulmonary infections, such as tuberculosis.

It is possible for one of the granulomatous diseases such as tuberculosis, sarcoidosis or one of the fungus diseases to give this clinical picture but they appear so unlikely to me that I will promptly discard them as possibilities—and hope that they will not be resurrected by the pathologist.

Now, assuming this is carcinoma of the lung, should the diagnosis have been made earlier (and if it had been made earlier could a life have been saved)?

Knowing the complete course of this patient's disease of course gives one a very definite advantage over those who were in charge of this patient at the beginning. In view of this infiltration on this first film taken in February 1950 and with the patient's family history of tuberculosis, I certainly would have

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made a diagnosis of minimal tuberculosis, probably active. At that stage of the disease, in view of the negative sputum studies, I believe close observation with monthly chest films and with cultures of sputum and gastric contents for T. B. would have been my way of handling this case. For 5 months the patient was watched closely with frequent chest x-rays.

The statement in the protocol states that the film in July 1950 showed no change. With this I differ as the lesion appears to me definitely more circumscribed and strongly suggestive of a mass rather than an infiltration. (Fig. I) A left lateral film I

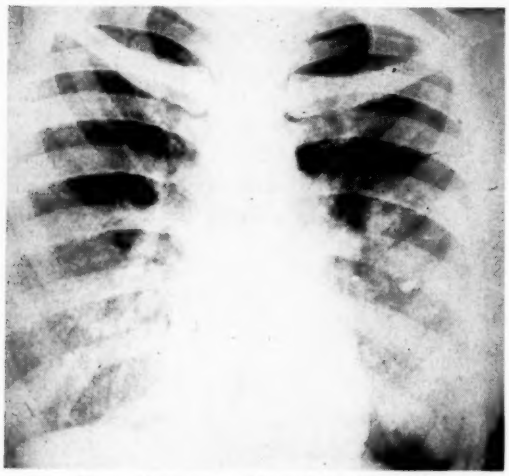

Fig. 1.—Appearance of lung lesion July 1950.

believe would have definitely established it as a mass. At this point I believe that hospitalization with bronchoscopy and further bacteriological studies should have been done. Since the lesion appeared as a mass, if tuberculous it would represent more than likely a tuberculoma and since this and a peripheral carcinoma of the lung would be the most likely diagnosis I would have had this patient explored at this time. In January 1951, 11 months after the first positive chest film, there was a definite increase in the pulmonary involvement by x-ray. The lesion appeared more definitely circumscribed and I believe the suspicion of malignancy should have been definitely aroused at that time and it probably was in the minds of those taking care of the patient. However, tuberculosis was still apparently the diagnosis of first choice of her physicians since pneumothorax was induced—although this is sometimes used as a diagnostic measure in determining the nature of a pulmonary lesion. The failure of the lesion to change either its size or its configuration following the induction of pneumothorax (Fig. II) should have

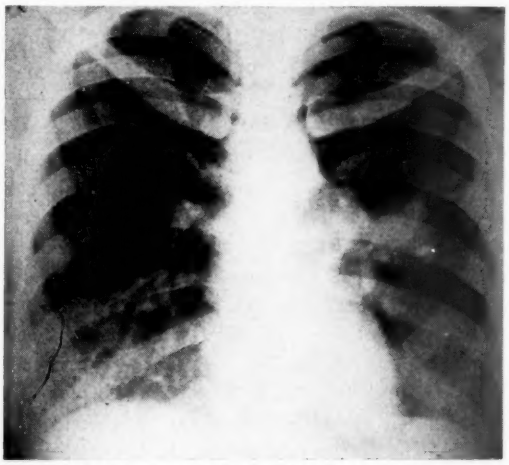

Fig. II.—Chest x-ray (April 1951) after pneumothoray.

further stimulated the suspicion of carcinoma and to the point where bronchoscopy and probably exploration would have been done. Certainly the supraclavicular lymphnode should have been removed. Even assuming the diagnosis of tuberculosis, the response of the lesion to pneumothorax indicated that this form of therapy would not be successful and that pulmonary resection was indicated.

Therefore, I believe that on the basis of the films of July 1950 and the subsequent ones exploratory thoracotomy was indicated. However, in view of the large hilar nodes which, in retrospect, were present on the film of February 1950, I doubt that eradication of the malignancy could have been accomplished if she had been explored following that film.

CLINICAL DIAGNOSIS:

1. Pulmonary tuberculosis
2. ? Multiple sclerosis

ANATOMICAL DIAGNOSIS:

1. Mucus—secreting adenocarcinoma of the lung with metastases to regional lymph nodes, adrenals, spleen and brain.

DR. RAY'S DIAGNOSIS:

1. Carcinoma of the lung with metastatis to brain.

PATHOLOGICAL DESCRIPTION (DR. W. G. REED) A6768—The body for autopsy was that of a thirty-six year old colored female, in fairly good nutritional status. The combined weight of the lungs was 500

grams. The left upper lobe bronchus was partially filled by a firm gray mass which was continuous with a more necrotic mass surrounding the bronchus. Multiple small tumor nodules were found in both lungs. The tracheo-bronchial lymph nodes were involved. The spleen contained a nodule, two centimeters in diameter. There was no gross or microscopic evidence of active tuberculosis. Although nothing was seen grossly in the adrenals, malignant cells were found microscopically. The brain contained multiple metastatic nodules, involving both gray and white matter, the basal ganglia bilaterially, and the ventricular system. The nodules ranged from 0.2 centimeter to 2.5 centimeters in diameter. All of them had glistening blue centers. (Fig. III)

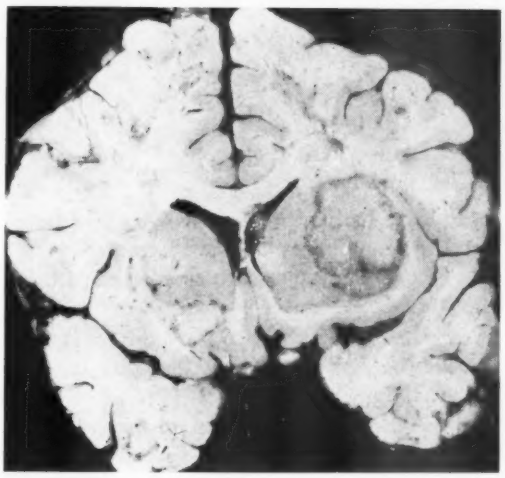

Fig. III.—Coronal section of the brain, showing the glistening metastatic nodules lying in the basal ganglia.

Microscopically, a relatively undifferentiated and pleomorphic adenocarcinoma was found originating in the lung. Frequently, it is a problem to decide whether these tumors arise from respiratory epithelium of the bronchi or from the mucus-secreting glands of the bronchial wall. In this case, no definite transition from normal respiratory epithelium to tumor epithelium could be found. Furthermore in several slides, bronchi are seen, with tumor pushing toward the lumen and destroying epithelium. This gives a distinct impression of origin from mucus-secreting glands. The tumor is arranged in bizarre and varying sized glandular structures, or as strands and small nests. The cells are moderately pleomorphic and show numbers of mitotic figures. Mucus production is not excessive.

The metastatic nodule in the spleen shows more of the papillary-cystic, mucus-producing characteristics. In the brain, the metastases form definite cystic structures, into the lumina of which project intricately branching papillary processes. (Fig. IV)

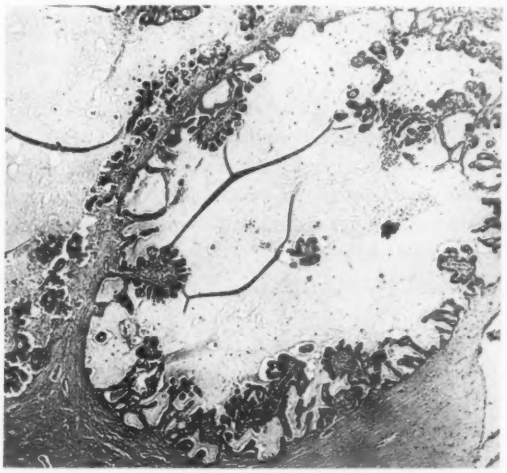

Fig. IV.—Microscopic view of the papillary cystadenomatous structure of the tumor of the brain.

These are lined by pleomorphic columnar epithelium, which has lost its polarity. Large quantities of mucus fill the cysts. This exemplifies the fact that metastatic lesions can take forms much different from the primary tumor. It may well be that the malignant cells formed cysts filled with mucus because the grain tissue offered less pressure resistance to their growth than did the lung.

Metastases to the brain from carcinoma of the lung are exceeded in frequency only by those from carcinoma of the breast.[1]

In summary, we have a case of mucus-secreting adenocarcinoma of the lung with metastases to the regional lymph nodes, adrenals, spleen, and brain.

BIBLIOGRAPHY

1. Willis, R. A.: The Spread of Tumours in the Human Body: p. 353.

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PUBLIC HEALTH

MACK I. SHANHOLTZ, M.D.
State Health Commissioner of Virginia.

Fluoridation of Public Water Supplies.

The Virginia State Department of Health approves and encourages the fluoridation of public water supplies for the partial control of tooth decay upon assurance that the community complies with the following conditions:

1. Endorsement by the local medical and dental groups concerned.
2. Approval by local health officials.
3. Evidence of public demand as shown by ordinances or resolutions by local governing bodies.
4. An application by the individual, firm, institution, organization or municipal corporation involved for a written permit and obtaining same before beginning fluoridation.
5. Development of plans, specifications, operating procedures and methods of supervision in conformity with requirements to be determined for each individual installation by the State Department of Health.

Two Virginia cities, Lynchburg and Charlottesville started adding fluoride to their water supplies last year. Blackstone, Fredericksburg, Fries, Norfolk, and Richmond have received their permits to begin fluoridation. There are approximately fifteen (15) other communities in the State that have started action towards fluoridation.

Virtually every health association in the country has gone on record favoring fluoridation for communities where water supplies are known to be deficient in fluoride. The House of Delegates of the American Dental Association in October, 1950, unanimously approved a resolution recommending the fluoridation of municipal water supplies to aid in the partial control of dental decay. The American Medical Association last year formally adopted a statement to the desirability and safety of fluoridation.

Dental and public health literature contains a large mass of evidence which demonstrates conclusively that there is approximately three times as much dental decay in children who use fluoride-free domestic water as there is in children who are born and reared in communities whose drinking water contains 1 part per million or more of fluorine. Controlled fluoride water studies conducted in Grand Rapids, Michigan; Newburgh, New York and Brantford, Ontario, Canada, started in 1945, report similar benefits to children from adding fluoride to their fluoride-free water supplies.

There has been some opposition to fluoridation, however. Most of this opposition has come from misinformed or uninformed persons who claim that fluoridation is "mass medication." Many of the arguments used are so obviously false that they need no answer. Competent research scientists, however, have carefully investigated and reported on many of these assertions. A summary of a few of these assertions and facts follows:

ASSERTION:

Fluorides in the public water supply may have harmful effects on human beings.

THE FACTS:

For generations over 3,000,000 persons in the United States have been living in areas where drinking water naturally contains fluorides in concentrations as high or higher than that recommended for dental health. Many studies have been conducted among these persons by competent investigators and the search has been painstaking, yet no one has been able to find any adverse physiological effect except an enamel defect known as dental fluorosis in areas where the fluoride concentration is several times higher than that recommended for dental health. Every national health group in the nation has attested to the safety of fluoridation.

ASSERTION:

Fluorine is a poisonous substance found in rat poison and insecticides.

THE FACTS:

In large amounts, fluorides are toxic both in humans and in animals. The recommended concentration in the fluoridation of water supplies, however, is neither toxic nor harmful. Chlorine is one of the most deadly gases ever known yet it is safely and effectively used in small amounts to purify drinking water. Many substances in common use by humans such as table salt and baking soda are beneficial when used in controlled amounts but may be poisonous when used in sufficiently large quantities.

ASSERTION:

Fluorides cause or accelerate the growth of cancer.

THE FACTS:

This misstatement results primarily from a mouse cancer experiment conducted at the University of Texas. An investigation of the experiment revealed that the mice under study were a special strain, highly inbred for susceptibility to mammary tumors. Approximately 16 to 100 per cent of these mice eventually succumb to mammary tumors regardless of their diet. Public Health Service investigators and officials of the National Cancer Institute reported that the experiment produced no evidence that there is any correlation at all between fluoridation and cancer development or growth.

ASSERTION:

Fluorides cause an unsightly staining of the teeth.

THE FACTS:

It is well known that high concentrations of fluorides in public water cause a dental fluorosis often called mottled enamel. When the water contains the recommended concentration of fluorides of one part per million, there is evidence of a very mild degree of dental fluorosis in less than ten per cent of children using such water. This degree of fluorosis does not cause staining. It is so slight that it can only be detected by careful dental examination. It usually appears on posterior teeth. It is much more desirable, dentally and socially, than carious teeth.

ASSERTION:

Fluorides will have a harmful effect on the kidneys of adults.

THE FACTS:

The Illinois State Board of Health has found no difference in the data on nephritis in different cities with varying amounts of fluorides in their public water supplies. Studies show that fluorides do not accumulate in the body. The ability of the body to eliminate fluorides is more than adequate for the amount ingested through fluoridated water.

ASSERTION:

Fluorides in the water make the bones brittle.

THE FACTS:

A detailed study of 1,458 high school boys in seven cities with varying concentrations of fluorides in public water supplies showed no effect on bone fracture experience. X-ray examinations of persons living in areas where the water contained from one to three parts per million of fluorides revealed no evidence of bone fluorosis. No relationship was found between fluorides and bone fractures or height and weight of individuals.

ASSERTION:

Fluoridation of public water supplies is "mass medication."

THE FACTS:

Fluoridation does not constitute medication under the accepted definition of the term any more than chlorination does. Fluoridation does not constitute a remedy. It does not treat an existing disease. Fluoridation supplies a normal constituent found in human teeth and makes fluorine available in the proper amounts for the development of decay-resistant teeth.

ASSERTION:

Fluoridation is "socialized medicine."

THE FACTS:

This obviously ridiculous charge indicated the character of many of the arguments used by opponents of fluoridation. Fluoridation of local water supplies is an accepted and proven measure to protect the dental health of all children. It is no more "socialized medicine" than is chlorination, immunization, pasteurization and the many other disease-preventing procedures in common use today.

ASSERTION:

Fluoridation only benefits children.

THE FACTS:

It is true that in the beginning of a fluoridation program, the greatest benefits will accrue to the younger children. They will continue to enjoy these health benefits throughout their adult life, however, and eventually the entire population of a community will be receiving full benefits from fluoridation. Studies among adults in fluoride areas show that the dental benefits continue throughout life.

MONTHLY MORBIDITY REPORT OF THE BUREAU OF COMMUNICABLE DISEASE CONTROL

	May 1952	May 1951	Jan.-May 1952	Jan.-May 1951
Brucellosis	2	7	9	26
Diarrhea and Dysentery	27	103	1,042	754
Diphtheria	8	11	44	63
Hepatitis	57	1	303	6
Measles	3,511	3,050	13,068	10,214
Meningitis (Meningococcal)	28	15	107	67
Poliomyelitis	0	1	10	15
Rabies in Animals	48	15	269	70
Rocky Mt. Spotted Fever	5	7	8	7
Scarlet fever	73	64	454	642
Tularemia	4	2	26	20
Typhoid and Paratyphoid	3	1	21	19

XUM

MENTAL HEALTH

JOSEPH E. BARRETT, M.D.,
Commissioner, Department of Mental Hygiene and Hospitals.

The Guidance Clinic as a Community Resource*.

Some forty years ago when guidance clinics first came into being they were established for a particular segment of the community—the delinquent child. The vision for their usefulness was very limited and they were developed as an adjunct of the Juvenile Court to provide a diagnostic service to assist the court in planning for the children who were in conflict with the law. Treatment was not considered to be a part of the clinic's function. Consequently, following the testing and psychiatric examination of the child, a report was furnished to the court, or else the parent was "told what was wrong with the child". Thus, referral to the guidance or psychiatric clinics in the early stages was largely involuntary on the part of the patient and his parents.

The studies and demonstrations by Sigmund Freud proving that early years are the most important in the formation of character traits opened the way for the preventive and corrective treatment of children, as well as adding to the skills of treating the problems of mental illness and adult adjustment. With this impetus given by Freud's work, and acting on the thesis that "childhood is a golden period for mental hygiene", such outstanding men as Dr. Adolph Meyer, Dr. E. E. Southard and others, and such organizations as the mental hygiene societies instigated and implemented the movement to develop guidance clinics with a wider usefulness to the community. They, along with others, recognized that diagnosis without treatment has a very limited usefulness, and that the clinic must take its place as one of the most important agencies in meeting one of the community's major concerns—mental health.

Along with the maturing and refining of treatment skills within the clinics there has been a steady emergence of new attitudes on the part of the public toward mental health, mental illness and emotional disturbance. There is more general acceptance on the part of the public that the person with these problems needs skilled help just as much as the individual with somatic illnesses needs his physician. Today, a smaller number of parents react as did one father when he was told the doctor advised that his son go to a guidance clinic. This father roared, "Does he think my boy is crazy? If he does, then he's the one that's crazy!" Nevertheless, much superstition still surrounds psychiatry and the guidance clinic, and many people still misunderstand what it means to go to a guidance center. There are others, however, who believe that there is something magical in the clinic's procedures and that once they get a child or an adult inside the clinic's door, all problems will be taken care of in a miraculous manner. This viewpoint does not take into account that there is a universal resistance to change, and that the mechanisms of rationalization and projection can hamper or block treatment, even though they are useful in making the client as comfortable as he is in his present situation. Thus, the skills of the clinic personnel are limited by the patient's desire for treatment and his recognition of the existence of problems. Furthermore, parents frequently have the attitude that they had no part in causing the child to be the way he is no more than they would have caused him to have a toothache. This, of course, is not true.

Let us consider the case of a 14-year-old boy referred to the clinic by his mother on the suggestion of the family doctor. The mother was told that the boy was "sissyfied" and needed help in making a personal and social adjustment. The mother, herself, saw no problem but was afraid not to come to the clinic because it had been recommended by the doctor. The case was accepted for study to determine whether the boy, since he was an adolescent, could be reached. The study revealed that he had good average intelligence, but that he was making a feminine identification, with definite homosexual trends; that he was quite neurotic and was already using somatic complaints as escape mechanisms. The boy himself rejected clinic treatment and denied the existence of any problems and the mother wanted nothing from the center, since she felt that her son

*Article prepared by Reynold D. Farris, Chief Psychiatric Social Worker, Roanoke Guidance Center, Roanoke, Virginia.

was like all other boys. She likes him the way he is. The clinic will continue to be blocked in meeting this boy's needs unless he becomes sufficiently uncomfortable to seek help independently of his mother.

Now let us consider the case of an 11-year-old girl referred to the clinic by her parents on the suggestion of the allergist. This very intellectually superior girl was rebellious, negativistic and reacted to emotional upsets and conflicts with her parents by having severe asthmatic attacks, frequently necessitating hospitalization. The doctor interpreted the emotional aspects of the child's illness to her parents who had already noted that the asthmatic attacks almost invariably followed scenes with her parents. They came to see that their relationship with and handling of the child was a major factor in her illness and were very eager for help. It can readily be seen that the prognosis for successful treatment in this and in similar cases is excellent.

Psychiatric sophistication is not required, but it is imperative that the patient or the parents of children be aware not only of the existence of a problem, but have a willingness to work toward its successful solution. Parents should have some awareness that they play a part in their child's life and because of that they, as well as the child, need help with the child's problems. The solution of the emotional conflicts of a child arising out of relionships within his family requires the simultaneous treatment of both child and parents. There is a teamwork approach to the treatment of children and best results are obtained only when someone interviews the parent while the therapist sees the child. The purpose of these interviews with the parent is to get him to observe himself and to discover the connection between how he feels and acts and how the child behaves. In most parent-child relationships a vicious circle has been set up. For instance, a mother's rejecting, hostile attitude not only contributes to a child's disturbances, but her attitude in turn aggravates the child's destructive and defiant behavior. Thus, successful treatment occurs when a change in attitudes is effected simultaneously in mother and child.

The guidance clinic can be of service in dealing with the problems of emotional and mental upsets that hinder the successful treatment of physical or social disorders with which the physician or others is concerned. Some of the many such instances would be the disturbed pregnant woman, the emotionally aggravated allergic condition, and the child who vomits when upset. This is, however, predicated on the basis that patients are prepared for coming to the guidance clinic and that there has been a proper evaluation of the patient's readiness and willingness for treatment.

The clinic also serves the community by providing a diagnostic service to physicians and agencies in cases where treatment is not indicated. This service is particularly useful in adoption and foster home placement cases. Diagnostic study is also useful to parents and others in planning for the exceptional child and for the child in need of some form of institutional care. Diagnostic service is limited by the demands made on a clinic for treatment service, since treatment is the main function of a guidance clinic. Diagnosis, in most cases, without adequate treatment facilities is relatively valueless, therefore, a clinic must be ready to give the treatment which the patient needs.

Good mental health is the guidance clinic's major concern and the clinic is the community's first bulwark of defense against mental illness and personal and social maladjustments. It achieves this purpose through its understanding of the dynamics of human behavior, and the utilization of the treatment skills of its staff in their teamwork approach to meeting the problems of people. The guidance clinic's unique contribution is that it uses the combined skills of the psychiatrist, psychologists and psychiatric social worker as a "team". However, the "team", as viewed by the clinic, includes not only the staff team but also the patient, his family, his physician and the community's institutions such as schools, churches, and social agencies. All of these working together are required for the promotion of good mental health within the community.

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MISCELLANEOUS

Industrial Medicine Program.

Dr. Charles L. Savage of Waynesboro, chairman of the sub-committee on Industrial Health of The Medical Society of Virginia, has called our attention to the following article which appeared in the May issue of Industrial Medicine and Surgery. It was reprinted from The Philadelphia Inquirer of November 25, 1951, and was written by Joseph F. Nolan, Medical Editor.

Preliminary moves with far-reaching implications for expansion of industrial medicine in this area have been made in a joint project of the Chamber of Commerce of Greater Philadelphia and the University of Pennsylvania. Announcement was made recently by Albert M. Greenfield, president of the chamber, and Dr. Harold E. Stassen, president of the University, that a joint committee has been studying the need for expansion of industrial medical service to combat on-the-job illness. With the realization that an immediate need for professional medical service must be met, the announcement said, a long-range program to aid business in establishing in-plant medical services has been developed to give professional attention to the on-the-job illness and thwart any trend toward socialized medicine. The committee reported: "A survey revealed that a great many doctors in private practice within the Philadelphia area devote part time to in-plant work. Also there are quite a number of physicians who are looking for part-time work in industry. All but a few of these physicians are inexperienced in industrial medicine and most of them have little conception of what they would do if a full-time job in a small plant were offered to them." As a result of this survey, a plan has been developed under which the University's School of Medicine will create a new division for industrial medical activities. The joint statement disclosed that the plan is one of both long-range and short-range objectives. The short-range plan will offer a post-graduate course in industrial health at the University. This course, although not an end to itself, will have a complete curriculum with qualified guest lecturers. Its purpose is to stimulate active interest in a coordinated program of industrial medicine for the Philadelphia area. The long-range program will integrate the industrial medicine viewpoint into under-graduate teaching; extend in-plant medical services to small plants, and maintain clinics to be established in the strategic areas to supplement the in-plant services of small plants by offering complete health services. Commenting on the University's plan, Greenfield said: "The Chamber of Commerce welcomes the opportunity to co-sponsor such an activity which promises new benefits to business as well as to the community at large. Modern business realizes its moral debt to employee's well-being, and business knows that healthy employees are more contented and less prone to absenteeism and avoidable accidents. The loss of full production in these days of emergency is curtailed by an adequate in-plant medical service." Greenfield said the chamber's Safety Council industrial health section would proceed to create further demand for industrial doctors by explaining the needs of industrial medicine to trade executives. The University's post-graduate course in industrial health will start early in 1952. The 12 afternoon classes will schedule pre-eminent medical and technical guest lecturers, specialists in industrial problems.

Planning a Summer Vacation?

Have you made all the necessary preparations?

While you are enjoying your vacation, remember we have a lot of our fellow countrymen fighting our battle in Korea. These fellows would like to be planning a vacation, too. You can help insure a future vacation for them . . . before you take yours.

Call your Red Cross for an appointment to donate a pint of blood for the armed forces before you leave. Your pint of blood is your gift of life to our fighting men. Plentiful supplies of plasma and blood for the wounded have helped to cut the death rate to about half the rate during World War II.

Make your appointment to save a life now—before you take that vacation. When you get the appointment, keep it. Our GI's keep their appointments in the foxholes of Korea each day. It is easier for you to keep yours at the blood center.

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WOMAN'S AUXILIARY TO THE MEDICAL SOCIETY OF VIRGINIA

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Auxiliary to the Richmond Academy of Medicine.

On May 16, 1952, Mrs. Maynard R. Emlaw was installed by Mrs. Henry W. Decker as President of the Woman's Auxiliary to the Richmond Academy of Medicine at a luncheon meeting held in the Academy Building. Mrs. Gilman R. Tyler was named president-elect and Mrs. William C. Barr was re-elected treasurer.

Other officers elected and installed at the meeting were: Mrs. Custis L. Coleman, vice-president; Mrs. G. Benjamin Carter, corresponding secretary, and Mrs. George K. Brooks, Jr., recording secretary.

New standing committee appointments were: Mrs. Edward S. Ray, "Bulletin" and "Today's Health"; Mrs. L. Benjamin Sheppard, parliamentarian and revisions; Mrs. Virgil R. May, historian; Mrs. Gilman R. Tyler, editorial; Mrs. Alfred L. Smith, legislative chairman; Mrs. Randolph H. Hoge, membership; Mrs. Custis L. Coleman, program, and Mrs. Fred L. Finch, public relations.

Appointments of the special committee chairmen were also made, as follows: Mrs. Hawes Campbell, devotional; Mrs. William F. Grigg, Doctor's Day; Mrs. John E. Stevens, personal relations; Mrs Hunter Jackson, volunteer work; Mrs. J. E. McGee, social, and Mrs. W. A. Dashiell, ways and means. Also named were Mrs. L. W. Hulley, Jr., yearbook; Mrs. R. B. Lawrence, nurse recruitment; Mrs. George Snead, civilian defense, and Mrs. J. David Markham, telephone.

A report was made at the meeting on the benefit tea for Sheltering Arms Hospital, given at "Windemere", the home of Dr. and Mrs. Douglas VanderHoof on May 9. This benefit tea is an annual affair and the entire proceeds are given to the Hospital; the amount of the check turned over to Mrs. William Frazier, president of the Board of Sheltering Arms, amounted to $777.00. Nurses from the senior classes of the Richmond hospitals were guests of the Auxiliary at the tea. In addition to its value as a philanthropic project, the tea always proves to be one of the highlights of the Auxiliary year from the sociability standpoint.

The next meeting of the Woman's Auxiliary to the Richmond Academy of Medicine will be held on September 19, 1952.

Northern Neck Auxiliary.

Mrs. Herman W. Farber, of Petersburg, President of the Woman's Auxiliary to The Medical Society of Virginia, was guest speaker at the Spring meeting of the Woman's Auxiliary to the Northern Neck Medical Association, at The Tides Inn, on Thursday, May 22nd. Mrs. Farber discussed the various objectives and committees of the Auxiliary.

Miss Hazel Nibb, of Richmond, Executive Secretary of the Graduate Nurses Association, was also a guest speaker. Miss Nibb spoke to the Auxiliary on "Nurse Recruitment".

The President, Mrs. J. Motley Booker, of Lottsburg, expressed the Auxiliary's pleasure in having Mrs. E. I. Lilly, of Baltimore, present at this meeting and welcomed as a new member, Mrs. Frank Pratt, of Palmer.

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BOOK ANNOUNCEMENTS

The Scalp in Health and Disease. By HOWARD T. BEHRMAN, A.B., M.D., Assistant Clinical Professor of Dermatology, New York University Post-Graduate Medical School; Adjunct Dermatologist, Mt. Sinai Hospital; Attending Dermatologist Hillside Psychiatric Institute; etc. 1952. The C. V. Mosby Company, St. Louis. 566 pages with 312 illustrations. Cloth. Price $12.50.

Bone Tumors. By LOUIS LICHTENSTEIN, M.D., Senior Pathologist, General Medical and Surgical Hospital, Veterans Administration Center, Los Angeles; Formerly Associate Pathologist, Hospital for Joint Diseases, New York; etc. 1952. The C. V. Mosby Company, St. Louis. 315 pages with 155 illustrations. Cloth. Price $10.50.

Principles of Refraction. By SYLVESTER JUDD BEACH, A.B., M.D., F.A.C.S., Consultant, Staff Main Eye and Ear Infirmary; Chief Ophthalmologist, Portland City Hospital; etc. 1952. The C. V. Mosby Company, St. Louis. 158 pages, illustrated. Cloth. Price $4.00.

Bacitracin. A Review and Digest of the Literature Up To and Including January 1952. Research Division, S. B. Penick & Company, New York. 127 pages.

Dynamic Psychiatry. Transvestism — Desire for Crippled Women. By LOUIS S. LONDON, M.D. Volume Two. Corinthian Publications, Inc., New York, N. Y. 1952. 129 pages, illustrated. Cloth. Price $2.50.

Technical Methods for the Technician. By ANSON LEE BROWN, B.A., M.D., Columbus, Ohio. Published by Anson L. Brown, Inc., Columbus, Ohio. 1950-51. xvi-784 pages. Illustrated. Cloth. Price $10.00.

The book contains chapters covering all fields of clinical pathology. Questions pertaining to each chapter may be well used for periodic checks of the knowledge of student-technologists. The book is an outgrowth of notes taken by Dr. Brown's students and is very useful in his laboratory; however, there was not sufficient effort put into conversion of these notes into a book which could be used by a large group of technologists. Many errors, some of them corrected after printing, make one think that others—some of them important—were not checked.

Methods used in Dr. Brown's laboratory should be brought up to date. They include obsolete tests like Esbach's method for detection of albumin in the urine, with three identical pictures of Esbach's test tube. Some tests are carelessly described and in the test for sugar in urine, Dr. Brown mentions that 8 drops of urine should be used, but does not determine the amount of Benedict's reagent added to the urine. Normal urine is erroneously described as not having any white, red blood cells and casts; however, we know that such exist in normal urine.

It is commendable that the origin of medical terms is given.

The description of blood cells is not accurate—monocytes do not have nucleoli as Dr. Brown mentions on page 173. The new nomenclature of blood cells is given verbatim as printed in an article in American Journal of Clinical Pathology in 1948. This nomenclature is still not accepted by all hematologists. The color plates with blood cells are very poor. Dr. Brown finds it necessary to include in this test many of his wise sayings and few of great people. This increases the impression that these are notes taken from his lectures. Some procedures are repeated verbatim—so is centrifugalization on page 24 and again on page 45. Many pieces of laboratory equipment are described unnecessarily in detail, giving even the color of the stand of the autoclave (page 48). The author describes in detail Leitz's photoelectric colorimeter, but does not explain the basic principles of spectrophotometry. There is no mention of flame photometry, which is probably not used in Dr. Brown's laboratory.

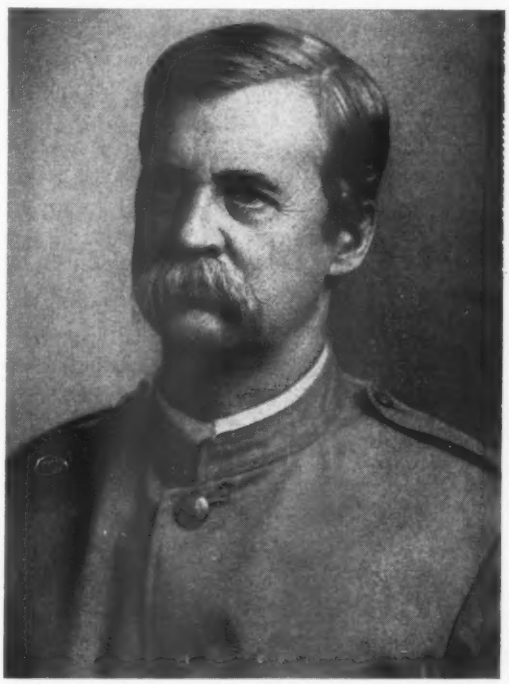
In conclusion, I do not think that Dr. Brown's book can stand the comparison with other books describing the techniques of laboratory tests. It should be limited to Dr. Brown's laboratory. The above mentioned shortcomings would make it hard to recommend this book to medical technologists.

H. G. K.

EDITORIAL

Henry Rose Carter, C.E., M.D. 1852-1925

IT IS remarkable how much of the important work in connection with the conquering of yellow fever was done by Virginians. Dr. Walter Reed, the chairman of the Yellow Fever Commission of the U.S. Army, was born in Gloucester County. Dr. Robert P. Cooke was one of the Commission's "human guinea pigs". He slept for twenty nights in the infected clothing building at Camp Lazear. It should be remembered that at the time, the "fomites" theory was still the most widely held theory and, while it was losing ground, it was necessary to prove or disprove it. At any rate it was a nauseating test. Dr. Cooke is still living in Rockbridge County, the only survivor of the experiments at Camp Lazear. General Gorgas, who put to a practical



Courtesy of Mr. Henry Carter Redd

HENRY ROSE CARTER, C.E., M.D.

test at Havana and at Panama Major Reed's findings, spent his boyhood in Richmond during the Civil War. Finally, there was Dr. Henry Rose Carter, who devoted all his professional life to sanitation in connection with yellow fever and malaria, and whose discovery of the "extrinsic incubation" period greatly facilitated the work of the Yellow Fever Commission.

In 1879, when Dr. Carter was sent to take charge of the epidemic in the Mississippi Valley, knowledge of yellow fever was meagre. It was not contagious in the sense that mere contact was all that was necessary to produce the disease. Some additional factor or factors were necessary. These in some way had to do with temperature, moisture, etc., and people talked of *miasma* and *fomites,* etc. Search was made in vain for

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causative bacteria and various organisms. How the unknown contagion was conveyed from patient to patient was a practical but unknown factor. In 1840, Dr. J. C. Nott advanced the idea that it was due to insects. Dr. Carlos Findlay, in 1881, incriminated the *Aedes aegypti,* upon epidemiological grounds. He tried some inoculation experiments to prove his theory but was unsuccessful. In spite of the great amount of work that was done, the situation remained confused. The officers of the U.S. Marine Hospital Service issued a report in 1898 which summed up the situation: "While yellow-fever is a communicable disease, it is not contagious in the ordinary acceptation of the term, but is spread by the infection of places and articles and bedding, clothing, and furniture. This is a process requiring several days (extrinsic infection), and during this period the yellow fever patient is as harmless as one suffering from a surgical complaint. . . . More recently the idea has been advanced that probably the germ of yellow fever enters the general circulation through the respiratory organs in some obscure manner, and incubating in the blocd directly poisons this life-giving stream. However this may be, the present opinion is that one has not to contend with an organism or germ which may be taken into the body in food or drink, but with an almost inexplicable poison so insidious in its approach and entrance that no trace is left behind."

When the epidemic at Memphis was over, Dr. Carter was put in charge of the yellow fever situation in the Gulf States. His first major task was to work out effective and simple quarantine regulations. A series of small scattered epidemics that occurred throughout the South was ideal for this work. According to Griffitts (South. M.J. **32**:841, 1939) Dr. Carter was well fitted for this task.

On February 26, 1901, Dr. Carter received the following letter from Dr. Reed:

"My dear Dr. Carter:

"Please accept my sincere thanks for the sentiments expressed in your kind letter of February 21st. I value highly your opinion of our work since I know of no one more competent to pass judgment on all that pertains to the subject of yellow fever. You must not forget that your own work in Mississippi did more to impress me with the importance of an intermediate host than everything else put together.

"With best wishes,

Sincerely yours,

WALTER REED"

Not only was he an accurate observer, but he kept records with mathematical accuracy. He was a Bible reading gentleman with model habits and was beloved by all with whom he worked. Consequently it was easy for him to get the state and local health officers to accept the quarantine regulations he had formulated. For the first time in history an epidemic of yellow fever in a favorable environment and in a non-immune population was stopped in mid summer. This happened at McHenry, Louisiana, in 1898. It was at this time that he discovered what he called extrinsic incubation. He soon noticed that a house did not become infectious until 12 to 18 days had elapsed after the first case had appeared. After secondary cases only four or five days were required. This difference Carter named extrinsic incubation. This observation removed an obstacle to accepting Findlay's theory of the mosquito transmission of the disease. In the meantime the Spanish-American War had been fought and won and Dr. Carter had been sent to Cuba as quarantine officer. Yellow fever appeared in the army of occupation. The time had come to settle definitely the etiology of the disease. A Commission was appointed under the leadership of Dr. Walter Reed and, with the aid of Dr. Carter's "extrinsic incubation", Dr. Findlay's mosquito theory was

soon proven by human inoculation, and the fomites theory disproved. The story is so well known that it is not necessary to go into details.

Dr. Carter continued his work in sanitation in connection with yellow fever and malaria in the United States Public Health Service and the International Health Board of the Rockfeller Foundation. His record with the United States Public Health Service runs as follows: assistant surgeon May, 1879; passed assistant surgeon July 1, 1882; surgeon February 1, 1892; senior surgeon, October 15, 1912; assistant surgeon-general by special Act of Congress March 4, 1915. In 1916 he was sent by the Rockefeller Foundation to Central and South America in charge of anti-malarial work and to Peru as advisor to the Peruvian Government in yellow fever prevention. Early in 1922, after his retirement from active duty in the United States Public Health Service, and at the advice of Dr. Wickliffe Rose, Director of the International Health Board of the Rockefeller Board and with material assistance from the Rockefeller Foundation, he began writing *The Early History of Yellow Fever.* He had practically completed this work when he died on September 14, 1925.

Dr. Henry Rose Carter was born at "Clifton" in Caroline County, August 25, 1852, the son of Henry Rose Carter and Emma Caroline (Coleman) Carter. When the future Doctor Carter was a few years old his parents moved to "North River" in Hanover County. The family was in comfortable circumstances until the Civil War rendered them destitute. They were refugees in Amherst County during the Civil War. His first schooling began at home. After the War he attended Aspin Hill Academy in Louisa County. He taught mathematics in an academy in Nelson County until he entered the University of Virginia where he graduated in engineering. Lameness, following a knee injury which was diagnosed as tuberculosis, was thought to preclude his following engineering. Johns Hopkins offered him a scholarship in mathematics, but he refused it because the incidental expense was too great. He taught mathematics in a boys' school in Baltimore and entered the Medical School of the University of Maryland, graduating in 1879. He entered practice with Dr. Charles Vest, a classmate, but they did not meet expenses and Dr. Carter took an examination for the Marine Hospital Service. He was commissioned assistant surgeon in 1879 and was stationed at Cambridge, Massachusetts, but by midsummer he was detached for yellow fever work in the Mississippi Valley. The rest of his life was devoted to the study of mosquitoes and their relation to yellow fever and malaria. He was recognized by his fellow workers all over the world as an authority and in 1904 Sir Ronald Ross proposed his name for the Nobel Prize in medicine.

Dr. Carter married Miss Laura Hook of St. Louis on September 29, 1880. They had three children: Laura Armistead (deceased) who married Admiral Stitt, Henry Rose Carter, Jr. of Birmingham, Alabama, and Edward (deceased). In the last five years of his life his heart condition reduced him to an elevator-cab existence with occasional visits to a hospital. He died of angina pectoris at his home in Washington, September 14, 1925, and was buried in Ashland, Virginia. A memorial service was held by the Norfolk County Medical Society and was addressed by Dr. B. M. Baber, Dr. Hugh S. Cumming, Dr. William S. Thayer of Johns Hopkins, Dr. George B. Young of the University of Virginia, Dr. Lomax Gwathmey, and Judge Thomas H. Willcox of Norfolk. These addresses were reported in the VIRGINIA MEDICAL MONTHLY (**53**:253, 1926).

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TRENDS IN MEDICAL EDUCATION

A GROWING dissatisfaction expressed by students and faculty alike with many phases of medical education has stimulated educators to reexamine the methods and policies currently employed in our medical schools.

The constant expansion of medical knowledge both in the basic and clinical sciences has led over a period of years to the organization of new and complex courses. This constant addition of courses without corresponding subtraction or rearrangement has resulted in a disorderly and oppressive medical curriculum, the value and practicability of which is now being challenged by medical educators. In many institutions the curriculum is being subjected to a thorough overhauling. Committees studying this problem have recommended condensation and better integration of some courses and elimination of others. The importance of teaching organ function in conjunction with structure has been stressed. Simultaneous teaching of the anatomy, physiology, pharmacology and pathology of a single organ or system of organs is being advised or actually attempted in some institutions. Determined interdepartmental efforts are being made to forge teams of basic science instructors and clinicians to teach important clinical problems.

In a talk given at a conference on Medical Teaching Techniques in Washington, D. C., Dr. William Parson of the University of Virginia discussed the need for teaching the teacher. He defined a good teacher as an individual with knowledge, enthusiasm, a capacity for communication, a sympathetic understanding of the student and a desire to assist his pupil to unlimited heights of achievement. He pointed out that too often teachers were chosen on a basis of their research accomplishments rather than their teaching qualifications. Nor is proficiency in the art of public speaking enough, for the teacher through his own personality must stimulate and direct the student.

Much criticism has been directed at the formal lecture. Undoubtedly, medical students are subjected to an unnecessary number of poorly organized and unstimulating lectures. However, the present trend to deemphasize the formal lecture has resulted as much from the deficiencies of the individual teacher as from the fundamental defects of this mode of teaching. Nevertheless, recognition of the inherent limitations of the formal lecture will lead to better balancing of the various teaching facilities available.

The effectiveness of the small conference group as a medium of teaching has long been recognized. The instructor utilizes group teaching to the best advantage when he encourages student participation. Group teaching requires a large and interested staff and the expenditure of a greater effort on the part of the faculty, but the dividends in medical education are correspondingly increased. In view of the personal sacrifices demanded of the conscientious teacher, there would seem to be no place on a medical school teaching staff for those who utilize their appointments for personal gain alone.

Undoubtedly visual aids in various forms have not been sufficiently exploited. Drawings, diagrams, photographs, charts and slides frequently provide an impressive medium for emphasizing certain facts. However, supplemental devices of this type can never substitute for the effectiveness of a good teacher.

At the University of California, under the guidance of Dr. Howard Bierman, an extensive study has been conducted during the past four years to analyze those processes of learning and retention which distinguish the good medical student from the average or poor one. Motivating forces figure prominently in the achievement of the individual. The problem of stimulating, guiding and maintaining these powerful motivating forces throughout the four years of medical school offers a real challenge

to the ingenuity and sympathy of the medical educator. The emotional background of the medical student plays an important role in determining his success or failure during his four years in medical school. Among other interesting facts unearthed in this study is the devastating role of the teacher who consciously or subconsciously antagonizes the students and thus establishes in his listeners a mental barricade against the permanent acquisition of information.

By no means the least important of the factors involved in the development of a good physician is the careful selection of the freshman medical student. Each individual dismissed from medical school may represent not only a failure of the student but also a failure of the admissions committee. Each applicant accepted constitutes a tremendous financial and social investment on the part of the medical school, and all means available must be employed in evaluating the inherent intellectual ability of the applicant as well as his psychological and emotional suitability for a medical career. A record of high scholastic achievement should not serve as the only yardstick for judging the potentialities of the applicant.

L. H. B.

SOCIETIES

Williamsburg-James City County Medical Society.

On March 24, following a social hour at the home of Dr. Granville L. Jones in Williamsburg, the members of the Society were the guests of the Eastern State Hospital for a steak dinner. After the business meeting was called to order by the President, Dr. Baxter I. Bell, miscellaneous business was considered which included the appointment of a Public Relations Committee: Dr. Ben Thomas Painter, Chairman, Dr. Granville L. Jones, both of Williamsburg, and Dr. E. B. Kilby of Toano.

Election of officers for the ensuing year was as follows: President, Granville L. Jones, M.D.; Vice President, Hugh G. Stokes, Jr., M.D.; Secretary-Treasurer, Frances E. Wood, M.D., all of Williamsburg.

The scientific session was a case presentation by Dr. Wendell J. Pile of Williamsburg. Dr. Pile discussed the difficulties in differential diagnosis in a case of avitaminosis and pellagra in which there had been a psychosis and unusual neurological findings.

On May 23 the Society members were the guests of Dr. and Mrs. E. B. Kilby at their home in Toano, for cocktails. The dinner was prepared by the ladies of the Hickory Neck Church and served at the Toano High School.

After the meeting was called to order by the President, Dr. Granville L. Jones, guests of the Society Dr. William Johns and Dr. J. Morrison Hutcheson, both of Richmond, were introduced. Dr. Frederik M. van den Branden who joined the staff of Eastern State Hospital on May 1, was introduced and elected to associate membership. Dr. van den Branden is a graduate of the University of Ghent, a specialist in Dermatology and Syphilology and was also inspector for the state insurance program in Belgium where he practiced before coming to the United States.

Various business matters were taken up including the appointment of Dr. Hugh G. Stokes, Jr., Williamsburg, as medical and technical advisor to the Williamsburg-James City County Ambulance Unit. The Ambulance Unit has done an excellent job in taking care of persons injured in highway accidents and other emergencies in this vicinity, and the Unit deserves to be complimented on its good work.

The guest speaker was Dr. J. Morrison Hutcheson of Richmond. Dr. Hutcheson gave an interesting and informative talk on the subject of hypertension. He discussed past and present ideas in regard to the nature and significance of blood pressure, evaluated some of the methods of treatment of hypertension, and outlined some of his own ideas as to how to manage the hypertensive patient with the knowledge and therapeutic methods at our disposal.

FRANCES E. WOOD, M.D., *Secretary*

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Wise County Medical Society.

On May 14, twenty-two enjoyed a dinner meeting and Dr. Leo N. Kirch's lecture on "Bladder Neck Obstructions", and Squibb's cinema on Tolserol. Three applications for membership will be voted on at our next meeting August 13th.

Dr. Geo. B. Setzler, Pennington Gap, who has continued his membership with us, was endorsed for re-appointment to the Virginia Board of Medical Examiners.

Our oldest member, Dr. R. W. Holley, Appalachia, is ill. Sympathy, greetings, confidence were conveyed to him and our president and others will visit him.

T. J. TUDOR, *Secretary*

The Medical Society of the Valley of Virginia

Held its Spring meeting on May the 22nd at the George Washington Hotel in Winchester, under the presidency of Dr. Charles L. Savage of Waynesboro. The program included the following papers:

Banti's Disease Treated Surgically—Dr. William C. Humphries, Woodstock

Trichinosis—Dr. Robert B. Gahagan, Clifton Forge

An Unusual Tumor in a Seven Year Old Girl—Dr. Karl Menk, Staunton

Infectious Polyneuritis—Dr. McKelden Smith, Staunton

The guest speaker was Dr. George P. Whitelaw, Assistant Professor of Surgery, Boston University School of Medicine, whose subject was The Surgical Treatment of Hypertension.

Officers of this Society are: President, Dr. Charles L. Savage, Waynesboro; vice-presidents, Drs. Lewis K. Woodward, Jr., Woodstock; Richard P. Bell, Jr., Staunton; and Dr. Harry G. Hudnall, Covington; secretary, Dr. McKelden Smith, Staunton; and treasurer, Dr. Harry G. Middlekauff, Weyers Cave.

Richmond Academy of Medicine.

Dr. Guy W. Horsley presided at the meeting of the Academy on May 27, at which time the following papers were presented:

Hormonal Treatment of Alcoholism (report of 265 consecutive episodes in 153 patients)—Dr. R. G. McAllister

Bleeding Due to Circulating Anticoagulants—Dr. Henry G. Kupfer.

At this time, the question was discussed as to instructing delegates to the State meeting as to the Academy's stand concerning the deletion of the word "white" in the constitution of the State Society.

Lynchburg Academy of Medicine.

The regular monthly meeting of the Academy was held on May 12th at the Lynchburg General Hospital.

The meeting was a symposium on "Cancer of the Body and Tail of the Pancreas". The program chairman, Dr. F. R. Whitehouse introduced the several speakers.

Dr. Brickhouse: "A General Outline of Cancer of the Body and Tail of the Pancreas".

Dr. Waters: "X-ray Findings".

Dr. L. R. O'Brian: "Conservative Surgical Treatment".

Dr. J. W. Devine, Jr.: "Radical Surgical Treatment".

Drs. Brownley, Craddock, John Hundley and Whitehouse presented a case each.

EDWIN A. HARPER, *Secretary.*

NEWS

Commencements of Medical Schools.

MEDICAL COLLEGE OF VIRGINIA

Entertainments incident to commencement, with alumni meetings and banquet, were held on June 1, 2 and 3. Preceding the final reception and dance, diplomas were awarded. The names of medical graduates with their hospital appointments are:

MEDICAL COLLEGE OF VIRGINIA HOSPITALS, Richmond—Drs. David Ware Branch, Richmond; William Carlyle Gill, Jr., Richmond; Carmen M. Jimenez Kaye, Mercedita, Puerto Rico; Thomas Pairo Overton, Richmond; John Stuart Prince, Stony Creek; Herbert Leon Ruben, Norfolk; William White Trigg, Jr., Petersburg; Herbert L. Weinberg, Suffolk; and Louis Reams Wilkerson, Raleigh, N. C.

JOHNSTON-WILLIS HOSPITAL, Richmond — Drs. John Fauntleroy Butterworth, III, Richmond; John William Gieseen, Radford; Emily Edwards Jones,

Smithfield; Thomas Roper Travis, Fredericksburg; and Robert William Wash, Jr., Pendleton.

STUART CIRCLE HOSPITAL, Richmond—Drs. Harvey Winfree Goode, Jr., Powhatan; and Peter Weaver Squire, Emporia.

DEPAUL HOSPITAL, Norfolk—Drs. William Cardwell Amos, Jr., Richmond; Earle Jerome Kerpelman, Richmond; Rose Marie Morecock, Richmond; George Oscar Shipp, Norfolk; and David Tyler, Richmond.

NORFOLK GENERAL HOSPITAL, Norfolk—Drs. William Marshall Atkins, Petersburg; Henry Vaughan Belcher, Norfolk; James Wendel Creef, South Norfolk; Jerome Stanley Gross, Norfolk; James B. Kegley, Jr., Waverly; Harvey Pretlow Rawls, Suffolk; James Royster Tarry, Brookneal; Phil Errington Trimmer, Jr., Richmond; Melvin Earl Yeamans, Richmond; and Phillip Clayton Yerby, III, Richmond.

RIVERSIDE HOSPITAL, Newport News—Drs. Elam Withrow Bosworth, II, Brownsburg; Keith Eugene Kinsey, Richmond; and Howard Jackson Maxwell, Lost Creek, W. Va.

WINCHESTER MEMORIAL HOSPITAL, Winchester—Dr. James Robert York, Stephens City.

LEWIS-GALE HOSPITAL, Roanoke—Drs. David Moomaw Brillhart, Troutville; Gene Edward Clapsaddle, Roanoke; Maury Claiborne Newton, Jr., Narrows; and Hubert George Tomlinson, Duffield.

U. S. NAVAL HOSPITAL, Portsmouth—Drs. Channing Lester Ewing, East Lake Weir, Fla.; Carroll Stanford Hamilton, Lynchburg; and Raymond Curtis Houghton, Portsmouth.

PHILADELPHIA GENERAL HOSPITAL, Philadelphia, Pa.—Drs. Benjamin N. Anderson, Jr., Hot Springs; Carey Jones Butler, St. Pauls, N.C.; Stanley Norman Cohen, Richmond; and Richard Ovid Rogers, Jr., Bluefield, W. Va.

MCKEESPORT HOSPITAL, McKeesport, Pa.—Drs. Asel Poe Hatfield, Harrisville, W. Va.; Ellis Franklin Maxey, Rustburg; and William Morris Riggins, Jr., Hampton.

U. S. NAVAL HOSPITAL, Bethesda, Md.—Drs. Richard Turberville Arnest, Jr., Hague; Thomas William Turner, Mouth of Wilson; and Frank Quinby Wingfield, Jr., Richmond.

UNION MEMORIAL HOSPITAL, Baltimore, Md.—Dr. Thomas A. E. Moseley, Jr., Richmond.

ST. MARY'S HOSPITAL, Huntington, W. Va.—Drs. George E. Arrington, Jr., Huntington, W. Va.; and Paul Raymond Kleykamp, Ashland, Ky.

CHARLESTON GENERAL HOSPITAL, Charleston, W. Va.—Drs. Roland Stillwell Birckhead, Glen Ferris, W. Va.; and Fred Lewis Goff, Charleston, W. Va.

OHIO VALLEY GENERAL HOSPITAL, Wheeling, W. Va.—Drs. Donald E. Fleming, Cove Station, W. Va.; and David Zackquill Morgan, Kingwood, W. Va.

MERCY HOSPITAL, Springfield, Ohio—Drs. David R. Brown, Buckhannon, W. Va.; Jean Plunkett Cavender, Buckhannon, W. Va.; Jerill Drexel Cavender, Charleston, W. Va.; Newman Allen Dyer, Charleston, W. Va.; John Alexander Murray, Richwood, W. Va.

SPRINGFIELD CITY HOSPITAL, Springfield, Ohio—Drs. Frank Alexander Hamilton, Jr., Martinsburg, W. Va.; Johnny Ray Hatfield, Gilbert, W. Va.; George Heltzel Hull, Durbin, W. Va.

CINCINNATI GENERAL HOSPITAL, Cincinnati, Ohio—Dr. William Benson McCutcheon, Jr., Durham, N.C.

UNIVERSITY HOSPITALS, Cleveland, Ohio—Dr. Lloyd Ulin Young, Falls Church.

JACKSON MEMORIAL HOSPITAL, Miami, Fla.—Drs. John Edward Bryant, Jr., Franklin; and Leonard Carroll Cantor, Richmond.

STATE OF WISCONSIN GENERAL HOSPITAL, Madison, Wis.—Drs. Robert Oliver Burns, Lebanon; and Ernest Dabney Shackelford, Jr., Richmond.

UNIVERSITY OF TEXAS MEDICAL BRANCH HOSPITALS, Galveston, Texas—Dr. Marshall Jennings Carper, Bluefield, W. Va.

BROOKE ARMY HOSPITAL, San Antonio, Texas—Drs. Powell Graham Fox, Jr., Raleigh, N.C.; and William Alexander Shelton, Keysville.

SCOTT AND WHITE MEMORIAL HOSPITAL, Temple, Texas—Dr. Richard Carl Snow, Hilton Village.

LOS ANGELES COUNTY HOSPITAL, Los Angeles, Calif.—Dr. Ann Shirley Carter, Richmond.

U. S. NAVAL HOSPITAL, San Diego, Calif.—Dr. Kenneth Darte Crippen, Alexandria.

ATLANTIC CITY HOSPITAL, Atlantic City, N. J.—Dr. John Speight Darden, Richmond.

GREENVILLE GENERAL HOSPITAL, Greenville, S.C.—Dr. Albert Pickett Dickson, III, Greensboro, N.C.

UNIVERSITY HOSPITAL, Augusta, Ga.—Drs. Robert Lester Gibson, Richmond; and William Edward Holladay, Jr., Gordonsville.

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GOOD SAMARITAN HOSPITAL, Phoenix, Ariz.—Dr. James Lester Grobe, Huntington, W. Va.

CENTRAL DISPENSARY AND EMERGENCY HOSPITAL, Washington, D.C.—Drs. Harry H. Howren, Jr., Richmond; and Jack Amory Lawson, Hampton.

GEORGE WASHINGTON UNIVERSITY HOSPITAL, Washington, D. C.—Dr. Robert Alan Morton, Portsmouth.

FAJARDO DISTRICT HOSPITAL, Fajardo, P. R.—Dr. Juan Francisco Jimenez, Santurce, P. R.

ST. JOSEPH'S MERCY HOSPITAL, Pontiac, Mich.—Drs. Carson Meade Keys, Nathans Creek, N.C.; and Harry Leroy Munson, Richmond.

EDWARD W. SPARROW HOSPITAL, Lansing, Mich.—Dr. Richard Wayne Wingfield Elkins, W. Va.

COOK COUNTY HOSPITAL, Chicago, Ill.—Dr. John Roger McDonough, Irwin.

MICHAEL REESE HOSPITAL, Chicago, Ill.—Dr. Bernard H. Miller, Norfolk.

KINGS COUNTY HOSPITAL, Brooklyn, N. Y.—Drs. Joseph B. Oram, Lester, W. Va.; and Frank Anthony Reda, Jr., Clarksburg, W. Va.

REX HOSPITAL, Raleigh, N.C.—Drs. Bernard Low Patterson, Roanoke; and George Franklin Tucker, Whitakers, N.C.

WORCESTER CITY HOSPITAL, Worcester, Mass.—Drs. Thomas Wirt Sale, Jr., Norfolk and William R. Tabor, Bluefield, W. Va.

UNIVERSITY OF OKLAHOMA HOSPITAL, Oklahoma City, Okla.—Dr. George Alexander Weimer, Bethany, W. Va.

Drs. Kenneth Martin Heatwole, Waynesboro and Eugene Bell Linton, Richmond were also given diplomas at this time.

UNIVERSITY OF VIRGINIA, DEPARTMENT OF MEDICINE

The final exercises for the 1952 class were held in conjunction with all other departments of the University, degrees being awarded and hospital appointments being announced as follows:

UNIVERSITY OF VIRGINIA HOSPITAL, Charlottesville—Drs. Milton Henry Brockmeyer, Pulaski; Charles Harper Crowder, Jr., South Hill; Jesse Wesley Cumbia, Broadnax; Meade Castleton Edmunds, Jr., Petersburg; James Watts Lipscomb, Huntington, W. Va.; Mary Jane Luke, Covington; Jason Eugene McCellan, Bristol; James Porter McNeil, Jr., Jacksonville, Fla.; William Clarke Pole, Hot Springs; William Norton Reingold, Norfolk; Beverly Lee Reynolds, Roanoke; and Joseph H. Smith, Newport News.

MEDICAL COLLEGE OF VIRGINIA HOSPITALS, Richmond—Dr. John Girardeau Murray, Jr., Greenville, S. C.

JOHNSTON-WILLIS HOSPITAL, Richmond—Dr. William Harvey Whitmore, Jr., Norfolk.

JEFFERSON HOSPITAL, Roanoke—Dr. Ben Lake Critzer, Pulaski.

DE PAUL HOSPITAL, Norfolk—Dr. Harold Louis Goldman, Norfolk.

NORFOLK GENERAL HOSPITAL, Norfolk—Dr. Fred Wasserman, Norfolk.

PORTSMOUTH NAVAL HOSPITAL, Portsmouth—Dr. George Edward Wire, Jr., Lovettsville.

MASSACHUSETTS MEMORIAL HOSPITALS, Boston—Drs. Kenneth William Berger, Norfolk, and Richard Hardaway Meade, III, Crozet.

PETER BENT BRIGHAM HOSPITAL, Boston, Mass.—Dr. Gustave Lennard Gold, New York City.

N.C. BAPTIST HOSPITAL, Winston-Salem, N.C.—Dr. Albert William Biggs, Memphis, Tenn.

WATTS HOSPITAL, Durham, N.C.—Dr. Donald Wood Glascock, Durham, N.C.

UNIVERSITY HOSPITAL, Chapel Hill, N.C.—Drs. James Eugene Grimes, Winston-Salem, N.C., and Nicholas Armistead Love, Raleigh, N.C.

CHARLOTTE MEMORIAL HOSPITAL, Charlotte, N.C.—Dr. Carter Ashton Sinclair, Bluefield, W. Va.

JOHNS HOPKINS HOSPITAL, Baltimore, Md.—Drs. Frank Stoddert Blanton, Farmville and Armistead Marshall Williams, Richmond.

SAN BERNARDINO COUNTY HOSPITAL, San Bernardino, Calif.—Dr. Loyd Warren Bond, Roanoke.

SOUTHERN PACIFIC GENERAL HOSPITAL, San Francisco, Calif.—Dr. Robert Mercier Hutt, Alexandria.

CEDARS OF LEBANON HOSPITAL, Los Angeles, Calif.—Dr. Robert Stanley Kaplan, Norfolk.

LETTERMAN GENERAL HOSPITAL (Army), San Francisco, Calif.—Dr. James Whitmell Ransone, Bowling Green.

PROVIDENCE HOSPITAL, Detroit, Mich.—Dr. Melvin Hugh Burke, Strasburg.

HENRY FORD HOSPITAL, Detroit, Mich.—Dr. Henry Page Mauck, Jr., Richmond.

OHIO VALLEY GENERAL HOSPITAL, Wheeling, W. Va.—Dr. Quintin Oswald Carr, Broadway.

THE NEW YORK HOSPITAL, New York City—Drs. Randolph Catlin, Jr., and Nelson Glasgow Richards, both of Charlottesville.

LENOX HILL HOSPITAL, New York City—Dr. Robert Earl McConnell, Bristol.

ST. LUKE'S HOSPITAL, New York City—Dr. Gilbert Fletcher Rieman, Virginia Beach.

MEADOWBROOK HOSPITAL, Hempstead, N. Y.—Dr. Jesse Reece Cover, Elkton.

KINGS COUNTY HOSPITAL, Brooklyn, N. Y.—Dr. Martin Paul Fischer, Brooklyn, N. Y.

HARRISBURG POLYCLINIC HOSPITAL, Harrisburg, Pa.—Dr. William Belfield Cave, Madison.

ABINGTON MEMORIAL HOSPITAL, Abington, Pa.—Dr. Richard Thomas Ellison, Jr., Philadelphia, Pa.

WILLIAMSPORT HOSPITAL, Williamsport, Pa.—Dr. William McLaurine Hall, III, Parkersburg, W. Va.

READING HOSPITAL, Reading, Pa.—Drs. James Edward John, Jr., Roanoke, and Carl Ellroy Stark, Alexandria.

PHILADELPHIA GENERAL HOSPITAL, Philadelphia, Pa.—Dr. John Lewis McCain, Southern Pines, N.C.

CINCINNATI GENERAL HOSPITAL, Cincinnati, O.—Drs. Nicholas Edward Davies, Clifton Forge; David Barnes Drewry, Drewryville; and James Bunting Kenley, Portsmouth.

ST. LUKE'S HOSPITAL, Cleveland, O.—Dr. Kenneth Hall Epple, Essex Falls, N. J.

UNIVERSITY HOSPITALS, Cleveland, O.—Dr. William Stuart Greenspon, Bluefield, W. Va.

CITY HOSPITAL, Cleveland, O.—Dr. John Edward OSBORNE, Miamisburg, O.

UNIVERSITY OF MINNESOTA HOSPITAL, Minneapolis—Drs. James Mitchell Fite, Muskogee, Okla., and Kenneth Stebbins Helenbolt, Medina, N. Y.

MEMORIAL HOSPITAL, Phoenix, Ariz.—Dr. Richard Orville Flynn, Phoenix, Ariz.

GALLINGER MUNICIPAL HOSPITAL, Washington, D. C.—Drs. Robert Humphreys Gruver, Washington, D. C.; Lionel Melvin Lieberman, Front Royal; and James Roger Smith, Ashland.

WALTER REED HOSPITAL (Army), Washington, D. C.—Drs. Harold Hubert Jeter, Jr., Florence, S.C., and Robert Basye Webb, Jr., Virginia Beach.

ST. ELIZABETH HOSPITAL, Washington, D. C.—Drs. Harvey David Karkus, Perth Amboy, N. J., and Aubrey Granville Tolley, Lynchburg.

ROYAL VICTORIA HOSPITAL, Montreal Canada—Dr. Euclid Murden Hanbury, Jr., Portsmouth.

JEFFERSON DAVIS HOSPITAL, Houston, Texas—Dr. Eugene Wilder Heatwole, Staunton.

UNIVERSITY OF TEXAS HOSPITALS, Galveston—Dr. Marion Moore Sherman, Hampton.

TRIPLER GENERAL HOSPITAL (Army), Honolulu, Hawaii—Dr. Holcombe Harris Hurt, Jr., Lynchburg.

SAN JUAN MUNICIPAL HOSPITAL, San Juan, Puerto Rico—Dr. Arturo Torres Machin, San Lorenzo, P. R.

MICHAEL REESE HOSPITAL, Chicago, Ill.—Dr. Edgar Elliott Peltz, Hampton.

MEMORIAL HOSPITAL, Wilmington, Del.—Drs. Hugh Walter Rule, Kingsport, Tenn., and Edward Ashby Woods, Jr., Pedlar Mills.

FT. SANDERS HOSPITAL, Knoxville, Tenn.—Dr. Charles Hallacy Spencer, Tunica, Miss.

Medical College of Virginia Alumni Association.

Meetings of the Alumni Association of the Medical College of Virginia were held under the presidency of E. Claiborne Robins of Richmond. Dr. H. Hudnall Ware of Richmond succeeded to the presidency and Dr. J. Asa Shield of Richmond was named president-elect. Mr. J. Curtiss Nottingham and Dr. Harvey B. Haag, both of Richmond, were re-elected secretary and treasurer, respectively. Miss Anne Skinner remains executive secretary. New members of the board of trustees elected are: Dr. James T. Tucker, Richmond; Dr. E. L. Alexander, Newport News; W. Roy Smith, Petersburg; Dr. Donald S. Daniel, Richmond; Dr. William N. Hodgkin, Warrenton; and Miss Frances Gordon, Richmond.

Announcement was made of the fact that two alumni would be awarded honorary degrees at the Commencement exercises on the 3rd: The degree of Master of Science in General Medicine to Dr. Walter C. Caudill of Pearisburg, a former President of The Medical Society of Virginia; and the degree of Master of Pharmacy to Mr. Ralph R. Rooke of Richmond.

The Association awarded special pins to the surviving members of the class of 1902 of the College and of the former University College of Medicine.

XUM

The Virginia Society of Ophthalmology and Otolaryngology

Held its annual Spring meeting at the Ingleside Hotel, Staunton, May 3, under the presidency of Dr. Robert H. Courtney of Richmond. Papers were presented by invitation by Dr. Conrad Behrens of New York on The Relationship of Some Ocular Disturbances to Reading Disabilities, and Joe Shinpaugh of the Virginia School for the Deaf and Blind at Staunton gave a Demonstration of Speech and Lip Reading, Language and Auditory Training as Taught to the Deaf Child.

After an intermission, the papers were presented by members as follows:

Errors in Management of Foreign Bodies of Food and Air Passages—Neil Callahan, M.D., Norfolk

Diagnosis of Intraocular Tumors—Edwin W. Burton, M.D., Charlottesville

The Management of Complications of Fenestration Surgery—Houston L. Bell, M.D., Roanoke

Following luncheon, a business session was held. Dr. A. D. Morgan of Norfolk succeeded to the presidency and the following were selected to serve with him: President-elect, Dr. Peter N. Pastore, Richmond; vice-president, Dr. M. K. Humphries, Jr., Charlottesville; secretary-treasurer, Dr. G. S. Fitz-Hugh (re-elected), Charlottesville.

The fall Clinical Session (Postgraduate) meeting will be in Charlottesville, November 18-1, 1952, and the spring meeting of the Society will be held with the West Virginia Academy of Ophthalmology and Otolaryngology on May 4 and 5, 1953, at Hot Springs, Virginia.

The West Virginia Academy of Ophthalmology and Otolaryngology

Held its fifth regular session at the Greenbrier, White Sulphur Springs, West Virginia, on May 12-13, 1952. The scientific program opened with Dr. Peter N. Pastore from the Medical College of Virginia, Richmond, who presented a paper on "Otitis Media and Mastoiditis Today". This was followed by a paper by Dr. Albert C. Esposito, Huntington, of "Malignant Melanomas of the Eye" and then Dr. Edward B. Holms of Parkersburg, presented a paper on the "Use of the Radium Pharyngeal Applicator in Office Practice."

On the second day the program started with Dr. John E. Bellows of Northwestern University, Chicago, who presented a paper on "Senile Changes in the Crystalline Lens." This was followed by a paper by Dr. J. A. B. Holt, Charleston, on "Plastic Procedures Included in Submucous Resection of the Nasal Septum", and the final paper was presented by Dr. Thomas Goodwin of Elkins, on "Why Use Mydriatics in Refraction."

The President-elect of the Virginia Academy of Ophthalmology and Otolaryngology was a guest at this meeting and he extended to the West Virginia Society an invitation for them to hold a combined session of both the Virginia and West Virginia Societies which will be held in Hot Springs, Virginia, the first week in May, 1953. This invitation was accepted by the West Virginia Academy.

News from the State Health Department

Dr. James M. Suter has been appointed Assistant Director of Local Health Services in charge of the Southwest District to succeed Dr. V. A. Turner.

Dr. R. W. Jessee, Health Officer who has been on scholarship status, has returned to duty and has been assigned to the Russell-Wise Health District with headquarters in Norton.

Dr. Ben Storer has been appointed Health Officer of the Smyth-Washington-Bristol Health District, and will assume his duties about July 1, 1952.

Dr. Charles M. Wylie, Health Officer who has been on educational leave, has returned and will begin his duties as Health Officer of Buena Vista-Lexington-Rockbridge Health District, effective July 1, 1952.

University of Virginia Department of Medicine News.

Dr. Fred H. Wilke, New York, class of '26 Medical Department, presided at the meeting of the Medical Alumni Association. Dr. J. William Hinton, class of '19, now professor of surgery and chairman of the department of surgery of the New York University Post-Graduate Medical School, gave the special address. Dr. Harry B. Taylor, who had spent forty-five years of his fifty since graduation as a medical missionary to China, was speaker at the medical alumni banquet on June the 6th.

Dr. Oliver B. Bobbitt has been appointed Director of the Clinical Laboratories of the University of Virginia Department of Medicine and Chairman of Clinical Pathology. A medical graduate of the

University in 1943, Dr. Bobbitt has served for the last five years with Dr. William Edward Bray, Professor of Clinical Pathology who retired July 1.

Dr. Arthur Ebbert, Jr., has been appointed Assistant to the Dean of the University of Virginia Department of Medicine and Instructor in Internal Medicine. A medical graduate in the University class of 1946, Dr. Ebbert served last year as Internal Medicine Resident.

Dr. Vincent Hollander, a member of the staff of the Sloane-Kettering Institute of the Memorial Cancer Center, New York City, will join the faculty of the University of Virginia Department of Medicine, January 1, 1953, as Assistant Professor of Internal Medicine and Coordinator of the Cancer Program.

Dr. Quentin Myrvik, Research Fellow in the Department of Microbiology of the University of Washington School of Medicine, has been appointed Assistant Professor of Microbiology at the University of Virginia Department of Medicine.

Three appointments to the faculty of Neurology and Psychiatry of the University of Virginia Department of Medicine are Dr. Charles A. Finnigan as Assistant Professor in charge of the new psychiatric out-patient clinic which was opened July 1, Dr. William D. Buxton and Dr. Cary Suter as Instructors.

The third Medicinal Chemistry Symposium of the American Chemical Society was held at the University of Virginia June 12-14.

Plans have been made for continuation of the post-graduate conferences for general physicians of the state at the University of Virginia. The conferences will be devoted to cardio-vascular disease, problems of infancy in general practice and diabetes.

Calendar of Meetings.

In this time of ever-increasing activity in medical organizations, the problem of conflicting meeting dates has become apparent. Often attendance at medical meetings is poor for no other reason than an allied group has unknowingly scheduled the same date.

Much of this confusion can easily be avoided. Immediately after a special meeting is planned, notify the State Society and the announcement will be published in the MONTHLY. On the other hand, always consult the MONTHLY before setting a date.

News must be received by the fifth day of any month to appear in the next month's edition.

Dr. M. B. Lamberth, Jr.,

Kilmarnock, has been elected president of the Lancaster County Lions Club for the coming year.

Dr. Montie L. Binder,

Internal medicine specialist in Newport News, has been named city and jail physician of that city, effective June 1, succeeding Dr. Thomas D. Morewitz, resigned.

Dr. William S. Sloan,

Petersburg, has been named as chairman of the American Red Cross Chapter of that city for the coming year.

Resign as Physicians with City of Richmond Service.

Dr. M. R. Emlaw, has resigned as part-time physician at City Home. Dr. George Benjamin Carter succeeds him.

Dr. Leonard D. Polikoff has resigned as medical examiner in charge of medical service for city employees. His place has been taken by Dr. Robert Miskimon who had been his assistant, and Dr. Richard Norton Baylor is the new appointee.

American College of Surgeons' Clinical Congress.

New surgical techniques and clinical developments will be presented at the 38th annual Clinical Congress of the College to be held in New York City September 22 to 26. More than 9,000 surgeons from all over the world are expected to attend the program of panel discussions, symposia, surgical forums, motion pictures, cine clinics, color television and exhibits. Headquarters will be at The Waldorf-Astoria. Dr. Frank Glenn, Surgeon-in-Chief, New York Hospital, is Chairman of the New York Committee on Arrangements.

Dr. Alton Ochsner of New Orleans, 1952 President of the American College of Surgeons, will preside at the opening evening session at which Dr. Harold L. Foss of Danville, Pennsylvania, will be installed as President for the year 1953. Dr. Evarts A. Graham of St. Louis is Chairman of the Board of Regents and Dr. Paul R. Hawley of Chicago is the Director.

XUM

Richmond Chapter, M.C.V. Alumni.

Dr. R. Campbell Manson was elected president of the local chapter, Medical College of Virginia Alumni at a meeting the middle of May. Wilhelm Haag was elected vice-president, and Dr. W. Yates League was named secretary-treasurer. Elected to the board of directors were Dr. W. Tyler Haynes, retiring president, Dr. E. Bowie Shephard, and Mrs. Jennie Caulkins.

Retirement of Two Veteran Salesmen, with Total of 87 years of Service, Announced by Parke, Davis & Company

Retirement of two veteran salesmen of Parke, Davis & Company, with a total of 87 years of service to the pharmaceutical firm, has been announced.

One of the men has served under six of the company's seven presidents. He is Alan Stuart, who joined Parke-Davis March 1, 1906, in the shipping department.

The other man has served under five Parke-Davis presidents. He is Frank G. Ebner, who started out as a salesman in Milwaukee September 1, 1910.

Harry J. Loynd, president, said both "have earned most happy and enjoyable periods of rest and retirement."

Dr. Raymond Brown,

Gloucester, has been named president of the Gloucester Lions Club for the coming year.

News from the Medical College of Virginia.

Commencement exercises closing the 115th session were held June 3 with 308 in the graduating classes: 102 in medicine; 48 in dentistry; 45 in pharmacy; 64 in nursing; 38 in physical therapy; 7 in hospital administration; three master of science degrees, and the first doctor of philosophy degree awarded by the college in its history. Dr. Walter C. Caudill of Pearisburg, former president of The Medical Society of Virginia, was awarded the honorary degree of Master of Science in General Medicine.

At the Commencement exercises it was announced that gifts and grants for the fiscal year 1951-52 thus far total $2,843,597.42. Included in this figure was the bequest of $2,250,000 from the late Adolph D. Williams of Richmond. Interest on about one and a half million dollars of the bequest is to be used for medical scholarships and fellowships; interest on the balance will be used for patients in the outpatient clinic. The outpatient clinic of the college by unanimous vote of the Board of Visitors of the college has been designated the A. D. Williams Memorial Clinic.

The college now has under construction: Randolph-Minor Hall, teaching unit and dormitory for nurses to be completed in July; cost equipped $687,000; the Wood Memorial Dental School Building, to be completed in 1953, approximate cost equipped $1,847,000. Funds are now available for a $250,000 addition to McGuire Hall and $1,875,000 for a hospital for the surgical treatment of Negro T. B. patients jointly with the State Board of Health.

Mr. C. P. Cardwell, Director of the Hospital Division, has been installed as president of the Virginia Hospital Association.

Dr. Harvey B. Haag, Professor of Pharmacology, is president-elect of the American Society for Pharmacology and Experimental Therapeutics.

Dr. R. Finley Gayle, Jr., Professor of Psychiatry and Neurology, has been elected secretary of the American Psychiatric Association for the third successive year.

The first Wortley F. Rudd Memorial Lecture honoring the late dean of the school of pharmacy was held May 15, with Dr. J. Harold Burn, Professor of Pharmacology, University of Oxford, as the guest lecturer.

Major faculty promotions effective July 1 are as follows:

Dr. Erling S. Hegre to Professor of Anatomy

Dr. H. Wallace Blanton to Associate Professor of Clinical Medicine

Dr. Edward S. Ray to Associate Professor of Medicine

Dr. William R. Kay to Assistant Professor of Clinical Medicine

Dr. Arthur Klein to Assistant Professor of Clinical Medicine

Dr. Howard McCue to Assistant Professor of Clinical Medicine

Dr. J. Warrick Thomas to Assistant Professor of Clinical Medicine

Dr. Charles E. Troland to Professor of Clinical Neurological Surgery

Dr. William Durwood Suggs to Associate Professor of Clinical Obstetrics

Dr. Edwin L. Kendig, Jr., to Assistant Professor of Pediatrics

Dr. Carolyn McCue to Assistant Professor of Pediatrics

Dr. Patrick H. Drewry, Jr., to Professor of Psychiatry

Dr. Merritt W. Foster, Jr., to Assistant Professor of Psychiatry

Dr. Lucy S. Hill to Assistant Professor of Psychiatry

Dr. A. Ray Dawson to Associate Professor of Clinical Physical Medicine and Rehabilitation

Dr. Edward M. Holmes, Jr., to Professor of Community Medicine

Dr. Paul W. Bowden to Associate Professor of Community Medicine

Dr. Hunter B. Frischkorn, Jr., to Assistant Professor of Clinical Radiology

Dr. George A. Welchons to Assistant Professor of Clinical Radiology

Dr. Saul Kay to Professor of Surgical Pathology

Dr. Leroy Smith to Associate Professor of Clinical Surgery

Dr. R. C. Siersema to Assistant Professor of Clinical Surgery

Dr. W. Glenn Reed to Assistant Professor of Pathology

Dr. M. Josiah Hoover to Associate Professor of Orthopedic Surgery

Miss Susanne Hirt to Associate Professor of Applied Anatomy

Miss M. Katharine Cary to Assistant Professor of Pathology

Miss Hazel Irvin to Assistant Professor of Clinical Pathology

Mrs. Louise Loving Jones to Assistant Professor of Anatomy

Dr. Alton D. Brashear to Professor of Anatomy

Dr. James E. McIver to Associate Professor of Denture Prosthesis

American Congress of Physical Medicine.

The 30th annual scientific and clinical session of the Congress will be held on August 25, 26, 27, 28 and 29, 1952 inclusive, at The Roosevelt Hotel, New York, N.Y. All sessions will be open to members of the medical profession in good standing with the American Medical Association. In addition to the scientific sessions, annual instruction seminars will be held. These lectures will be open to physicians as well as to therapists, who are registered with the American Registry of Physical Therapists or the American Occupational Therapy Association.

Full information may be obtained by writing to the American Congress of Physical Medicine, 30 North Michigan Avenue, Chicago 2, Illinois.

Added to Sandoz Field Staff.

Sandoz Pharmaceuticals announces the addition of several field representatives who have completed their training and are now in their new territories. Mr. Charles Halloran is the one who will have headquarters in Richmond and cover Richmond and the surrounding area.

Postgraduate Gastroenterology Course.

The National Gastroenterological Association announces that its Fourth Annual Course in Postgraduate Gastroenterology will be given at the Hotel Statler in New York City on 23, 24, 25 October 1952.

As in past years the Course will again be under the direction and co-chairmanship of Dr. Owen H. Wangensteen, Professor of Surgery of the University of Minnesota Medical School, who will serve as surgical co-ordinator and Dr. I. Snapper, Director of Medical Education of The Mt. Sinai Hospital, N. Y., N. Y., who will serve as medical co-ordinator. They will be assisted by a distinguished faculty selected from the medical schools in and around New York City.

One complete session will be devoted to a Clinical Pathological Conference at the Mt. Sinai Hospital in New York City.

For further information and enrollment write to the National Gastroenterological Association, Department GSJ, 1819 Broadway, New York 23, N. Y.

The New Medical Center at Louisa.

A combination health and medical Center, was dedicated on June the 15th. One wing of the one-story building will be occupied by the Louisa County Health Department and the basement will have office space for physicians and a dentist. The Center will have twenty-three beds, including an isolation ward, a delivery room, and emergency operating facilities. The Center's staff will be composed of Drs. H. W. Judd, E. B. Pendleton, H. S. Daniel, John W. Barnard, T. E. Stanley, Griffith B. Daniel, Evelyn P. Daniel, M. T. Lowry, Stuart W. Selden, and Charles K. Perkins, dentist.

XUM

Medical Resident Wanted.

Beginning July 1, fully approved 165-bed general hospital has opening for Medical Resident. Stipend $150 a month and maintenance. Address "Medical Director" C. & O. Hospital, Huntington, W. Va. (*Adv.*)

For Sale—

Wappler Jr. Vertical Fluoroscope, Serial #30583—$150.00; G.E. Model "B" Electrocardiograph, Serial #128920, Type (2)—$25.00; One very tall heat lamp—$10.00; All are in perfect condition with a lot of good film for E.K.G.

May be seen by contacting Miss Imogen Reynolds, Caldwell, West Virginia. (*Adv.*)

Wanted—

Experienced Superintendent of Nurses and Laboratory Technician for a small hospital. A satisfactory salary with maintenance. Lebanon General Hospital, Lebanon, Va. (*Adv.*)

OBITUARIES

Dr. Edmund Pendleton Tompkins,

Prominent in medical circles in his section and an enthusiatstic researcher and recorder of the history of Rockbridge County, died May 26 after a short illness. He was 83 years of age and a graduate of the Medical College of Virginia in 1897. He began practice in Rockbridge County but, after a post-graduate course, located in Roanoke and while there served a term as president of the Roanoke Academy of Medicine. In 1925 he located in Lexington and had practiced there until his 82nd year. He was coroner of Rockbridge County for many years and founded and was active in the Rockbridge Historical Society. He had for many years been a member of The Medical Society of Virginia in which he also held Life membership. He is survived by his wife and two sons, doctors who are located in San Francisco and in Oklahoma City.

Resolutions on the Death of Dr. William S. Snead

Dr. William S. Snead, whose death occurred on April 3, 1952, was one of the prominent practitioners of Newport News. He was born in Spotsylvania County February 8, 1886.

After early education in local schools, he attended the Oakland Academy for a year and then William and Mary College for three years. His father's death in 1906 caused the lapse of a year. He entered the Medical College of Virginia in 1907 and graduated in 1911. He practiced medicine in Lunenburg County in 1918 prior to coming to Newport News, Virginia in March of that year, where he practiced until his death.

Dr. Snead had been associated with the Riverside Hospital since coming to Newport News and was on the surgical staff.

In May, 1908 he married Miss Marie Gordon Morris of Newport News. They had three children, two sons and a daughter, who survive him.

WHEREAS, Newport News has lost an outstanding physician and a member of this community,

BE IT RESOLVED: That these resolutions be entered in the minutes of the Medical Staff of the Riverside Hospital, and a copy of same be sent to the VIRGINIA MEDICAL MONTHLY, and to the family.

Committee:

WILLIAM O. POINDEXTER
J. HUGHES MABRY
EDWARD B. MEWBORNE, *Chairman*

Dr. Elton A. Ratcliffe,

Richmond, died May 30th, after having been in bad health for sometime. He was sixty-three years of age and a graduate of the Medical College of Virginia in the class of 1915. He was a Mason and a member of The Medical Society of Virginia. His wife and a daughter survive him.

Dr. Travis Barton Twyman

Died May 14th at his home at Locust Dale. He was sixty-five years of age and had graduated from the Medical College of Virginia in 1908. He had not been engaged in active practice for sometime. His burial was with Masonic rites. Two sons survive him.